MEDICAL LIBRARY

DEC 2 1947

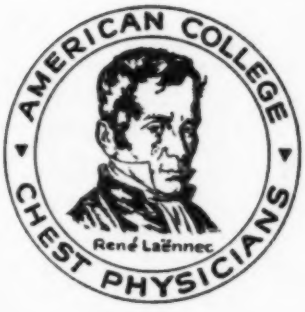
VOLUME XIII NUMBER 6

DISEASES

of the

CHEST

OFFICIAL PUBLICATION



PUBLISHED BI-MONTHLY

NOVEMBER - DECEMBER
1947

PUBLICATION OFFICE, ALAMOGORDO ROAD, EL PASO, TEXAS
EXECUTIVE OFFICE, 500 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS

Entered as Second Class Matter August 18, 1936, at the Postoffice at El Paso, Texas
Under the Act of Congress of August 12, 1912.

Copyright, 1947, by the American College of Chest Physicians

BUY CHRISTMAS SEALS — FIGHT TUBERCULOSIS

Christmas Seals

. . . Your Protection
Against Tuberculosis

Where the science of treatment is first

ROCKY GLEN SANATORIUM McCONNELSVILLE, OHIO

FOR THE MEDICAL AND SURGICAL TREATMENT OF TUBERCULOSIS

LOUIS MARK, M.D., Medical Director, 677 North High Street, Columbus, Ohio

HARRY MARK, Superintendent **MRS. H. A. PHILLIPS,** Asst. Superintendent

FRANK LANDE, M.D.
Resident Medical Director

HENRY BACHMAN, M.D.
Consultant

Beautiful Surroundings **Graduate Nurses** **Reasonable Rates**

THE CALIFORNIA SANATORIUM

BELMONT, CALIFORNIA

Located in the well-known sunny belt of the Peninsula, about thirty miles south of San Francisco. Large park, semi-tropical grounds, walks, especially laid out for graduated exercise.

Not too hot in summer — not too cold in winter.

Physicians on duty day and night — Graduate nurses.

THOMAS B. WIPER, M.D., Director and Consultant in Thoracic Surgery
W. N. TORRE, M.D., Resident Clinician

SAN FRANCISCO OFFICE: 450 SUTTER STREET PHONE: DOUGLAS 5793

DISEASES *of the* CHEST

VOLUME XIII NOVEMBER-DECEMBER, 1947 NUMBER 6

Multiple Segmental Resection in the Treatment of Bronchiectasis

RICHARD H. OVERHOLT, M.D., F.C.C.P.,*
REEVE H. BETTS, M.D., F.C.C.P.* and
FRANCIS M. WOODS, M.D.
Boston, Massachusetts

Chest specialists now recognize that bronchiectasis is a disease which in most cases is best treated by surgery. Many reports, especially those of Perry and King,[7] and Bradshaw, Putney and Clerf,[2] have emphasized the seriousness of bronchiectasis untreated by excision. They have shown that on the average patients having symptoms since early childhood do not live much beyond the age of 30 and that life expectancy after the development of symptoms is only 13.5 years in the fatal cases. Bradshaw, et al[2] further found that in the fatal cases the duration from diagnosis to death was only 1.8 years. This came as a surprise to many physicians, who, although they recognized that the patient with bronchiectasis had his "ups and downs," assumed that most of them went on for an indefinite time without very much change in their condition. An appreciation of the fallacy of this assumption has provided an impetus to the perfection of surgical treatment. Furthermore, the introduction of chemotherapy and the antibiotic drugs has increased rather than decreased the need for excisional therapy. Many patients who would have otherwise succumbed to acute exacerbations are now reconverted to the chronic form of the disease. The chest specialist, therefore, is faced with a problem of caring for more patients who live and suffer longer with the disease.

The changes that have taken place in the surgery of bronchiectasis during the past few years have been outstanding. There

*From the Department of Surgery, Tufts College Medical School, and the New England Deaconess Hospital, Boston, Massachusetts. Presented at the 13th Annual Meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 6, 1947.

Copyright, 1947, by the American College of Chest Physicians.

is little resemblance between the precise dissection and excision of pulmonary tissue as carried out today and the comparatively crude lobectomy by the tourniquet method of a few years past. Gone is the two-stage operation and the prolonged periods of hospitalization. Postoperative bronchial fistulae and empyema instead of being common sequelae are rarely encountered. Formerly one expected a few days to a week of rather profound prostration and critical illness. Now the postoperative course is short, benign, and, surprisingly, not very uncomfortable. What are the factors responsible for these gratifying changes? What are the prospects for further improvement and refinement in the surgical treatment of this disease? Recent experiences and developments which answer these questions will be presented.

In the beginning it was essential that a technique be developed which would result in an acceptable mortality for resection of the simplest units of excision,—a pulmonary lobe or a lung. The dissection technique used in dissecting the hilum of the lobe, now in almost universal practice, has proved to be highly successful. First reports of a reduction to a 3 or 4 per cent mortality have been superceded by others of sizable groups of cases (100 or over) with no mortality or a mortality as low as 1 per cent. Such figures, however, apply mainly to uncomplicated cases with well-localized disease since surgical treatment has been limited in its application by and large to patients suffering from unilateral disease.

It is well known, however, that bronchiectasis is not necessarily a lobar disease. Churchill and Belsey[3] have called attention to the frequency with which the lingular segment of the left upper lobe is involved in cases of bronchiectasis. They also pointed out that frequently the superior segment of the lower lobe is free of disease when basal segments are involved. Blades,[1] and Clagett and Deterling[4] have also emphasized the fact that bronchiectasis may involve only segments and have described a clamp and suture method for removing portions of lung of less magnitude than a pulmonary lobe. In our own previously reported experience with bronchiectasis,[6] it was found that in only 15 per cent of the cases was an entire lobe involved. In the majority of cases the disease was found to be multi-segmental. In approximately 30 per cent the involvement was bilateral.

Segmental Localization

Roentgenography and bronchography are the basis of accurate pulmonary localization. The value of fluoroscopy and a study of roentgenograms taken in various projections is well known. An appreciation of the necessity for complete bronchographic study of all bronchopulmonary segments is of more recent development.

For the bronchiectasis patient, an intelligent plan of treatment cannot be outlined without accurate knowledge of the condition of all the segmental bronchi. Simply injecting the opaque medium and positioning the patient is not sufficient as frequently some segments will not fill adequately by such a method. We believe a preferable plan is to do the actual injection of the contrast material under fluoroscopic control in order that the segments can be visualized and filled with certainty. The bronchi of both lungs may be mapped out at the same time if roentgenograms in the oblique projections are taken. The oblique position separates the bronchi and prevents overlapping of their images.

A number of acceptable and workable systems of nomenclature for the bronchopulmonary segments have been devised. Jackson and Huber[5] have named the ten basic bronchopulmonary segments of each lung according to their position within the lobe. We have chosen to use their nomenclature (Figure 1). The right and left lungs do not differ greatly. The lingular segment of the left upper lobe is homologous to the right middle lobe. Other changes in the pattern of the segments in the left upper and lower lobes on the left result from a combining of two segments of the upper (apico-posterior) and two segments of the lower lobe (antero-medial).

The main advantage of any of these newer systems of nomenclature is the ability thereby to discuss more accurately anatomical divisions of less than lobal area. With the availability of a precise method of definition, more detailed study and appraisal

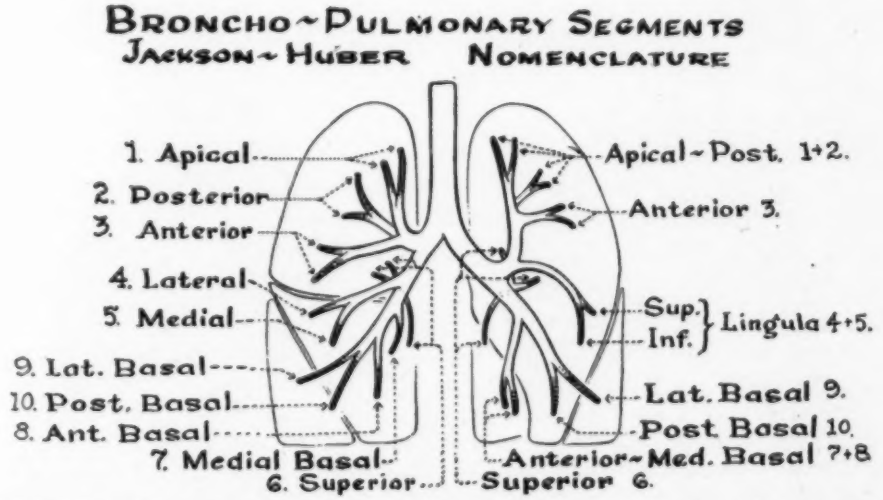


FIGURE 1: Diagram of the 18 bronchopulmonary segments and their names suggested by Jackson and Huber. This nomenclature is based upon the position of the segment within each lobe. On the left side there are eight instead of ten segments. The left apical and posterior segments have a common trunk. Likewise the anterior and medial basal segments are best considered as one surgical unit because of a common origin of their segmental bronchi.

of disease processes has resulted. It has been generally appreciated for some time that pulmonary abscesses, for instance, are frequently situated in the upper posterior part of the lower lobe (superior segment) or in the anterior portion of the upper lobe (anterior segment). The explanation for this is obvious when one appreciates the anatomy of the segmental bronchi supplying these areas. When a patient is in the supine position, the superior divisional bronchus of the lower lobe is directly dependent. The same is true of the anterior segmental bronchus of the upper lobe with the patient in the lateral decubitus position. Thus material from the naso-pharynx or other segments of the lung will find its way into these dependent areas by gravity.

Bronchiectasis likewise shows a predilection for certain segments. Most frequently involved are the basal segments of the lower lobes, the left more often than the right.[6] Quite commonly

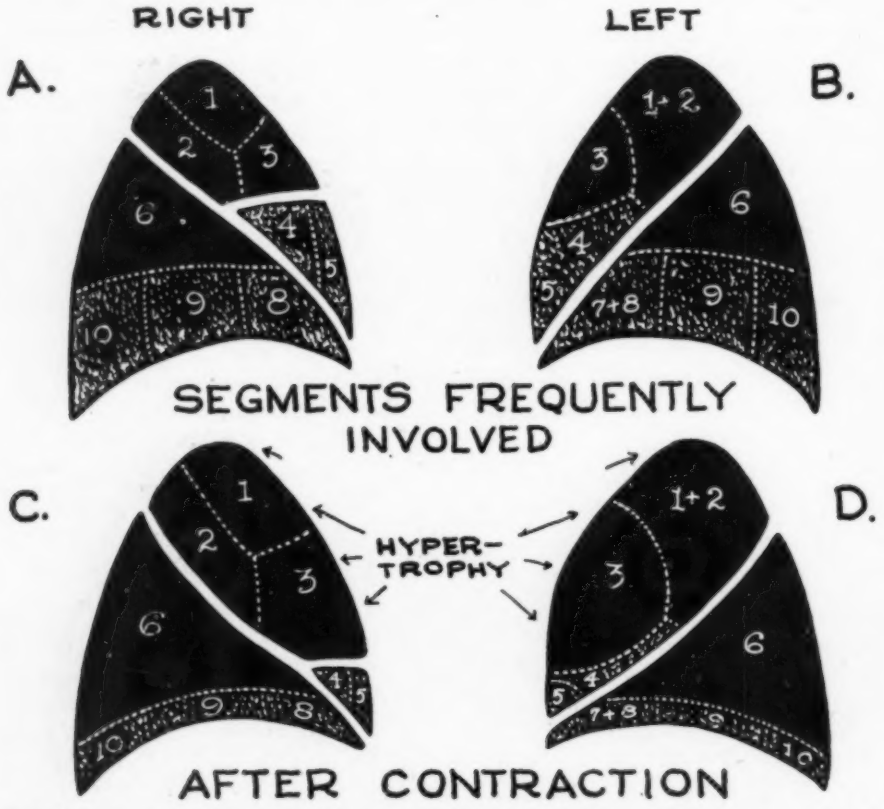


FIGURE 2: Diagrams of the lateral surface of the lungs with surface area of the segments outlined. The stippled areas of Diagrams "A" and "B" indicate those segments most frequently involved in bilateral bronchiectasis. Diagrams "C" and "D" show relative areas after the diseased segments have contracted and the healthy segments have enlarged. If the disease originated during the developmental period, the enlargement of the healthy segments is accomplished by true growth or hypertrophy. If the volume change comes later in life, the uninvolved segments enlarge by simple over-expansion (emphysema).

the lingular portion of the left upper lobe is affected, occasionally by itself but usually in conjunction with the basal segment of the left lower lobe. On the right side the homologous areas are most generally involved, that is, the basal segment of the lower lobe and the middle lobe (Figure 2). Only rarely are other portions of the lung the seat of bronchiectatic changes except when associated with, or as sequelae of, preceding infectious processes. For instance, one occasionally sees bronchiectasis limited to the superior division of the lower lobe following a lung abscess in the same location.

The knowledge that bronchiectasis is usually segmental, rather than lobar, in its distribution stimulated our interest and that of others in the pulmonary segment as a possible unit of excision.

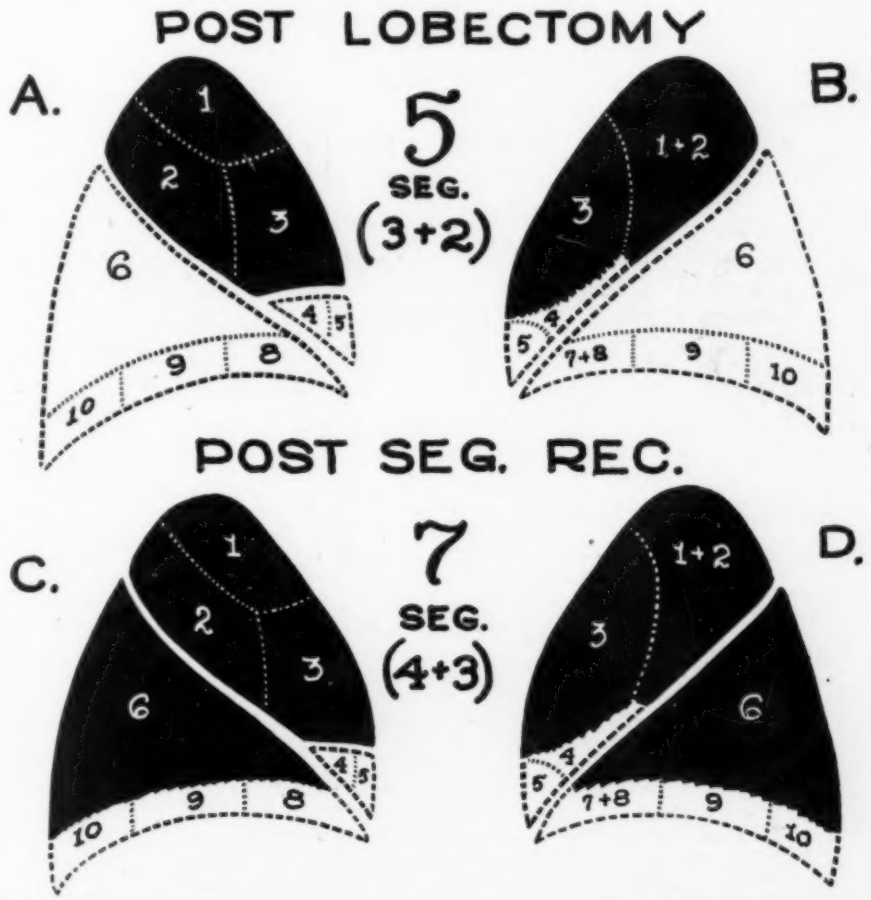


FIGURE 3: Diagrams of the lateral surface of the lungs with the segmental surface areas outlined. Diagrams "A" and "B" show small remaining areas if lobes are used as the units of excision in bilateral bronchiectasis. See Figure 2 for illustration of the most common distribution of bilateral disease. Diagrams "C" and "D" show relative areas conserved by using the segment as the unit of excision in bilateral disease.

Much credit should go to Churchill and Belsey,[3] to Blades,[1] and to Clagett and Deterling[4] for their concept of the problem and for their contributions in the development of the technique of segmental resection. One of the goals in excisional therapy is to eradicate all the disease without sacrificing any normal tissue. If this goal is to be approached in bronchiectasis, segments rather

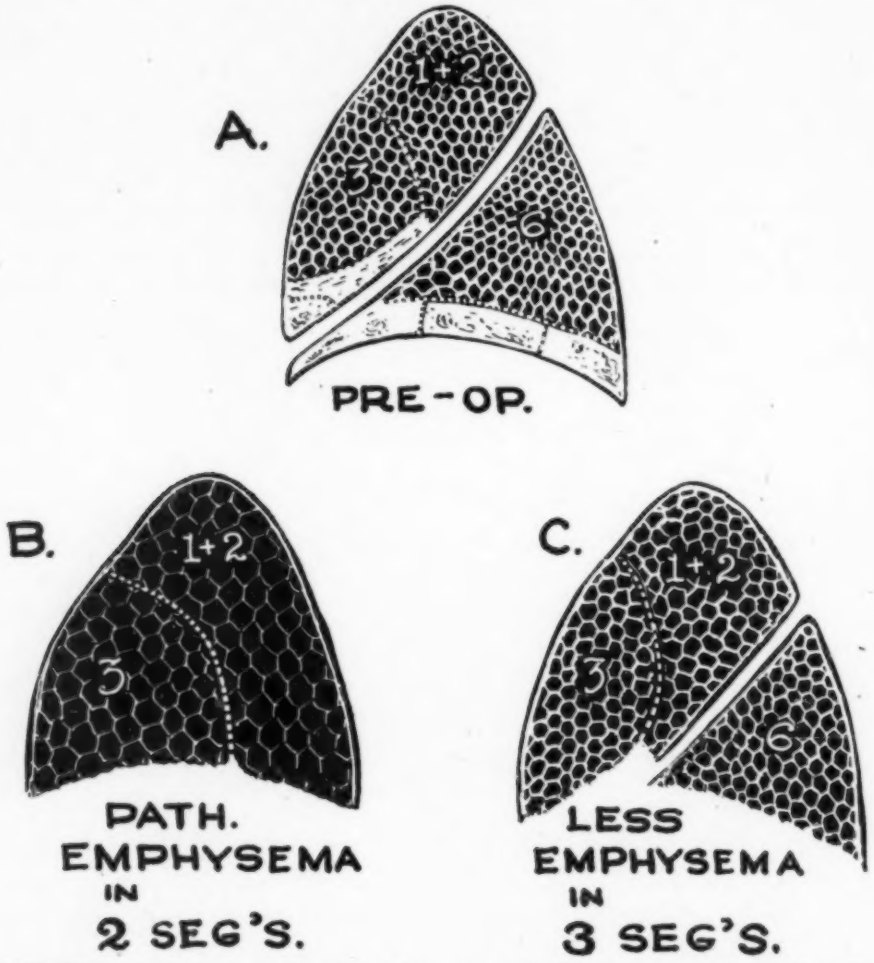


FIGURE 4: Diagrams of the lateral surface of the left lung illustrating the advantage of employing the segment rather than the lobe in bronchiectasis in adults. Diagram "A" shows the relative size of the segments in a case of bronchiectasis of the lingular and basal segments. The three remaining healthy segments have hypertrophied because the patient developed bronchiectasis early in childhood. As the disease contracted the involved segments, the upper segments of each lobe grew to form abnormally large segments. If surgical treatment is carried out after puberty, any further expansion will take place by a process of simple over-stretching. Diagram "B" shows the over-expansion of the remaining two segments if the entire lower lobe is removed together with the lingula. The resulting emphysema may be pathological. Diagram "C" shows the lateral surface of the left lung after segmental resection for basal and lingular bronchiectasis. The previously hypertrophied superior segment of the lower lobe has been preserved. Its presence greatly lessens the need of the remaining two segments of the upper lobe of undergoing over-expansion.

than lobes become the excisional units for most cases. Furthermore, the conservation of all healthy pulmonary tissue becomes a necessity in treating many patients with extensive bilateral disease (Figures 3 and 4). The marked pulmonary insufficiency of the patient following removal of both lower lobes, the right middle lobe, and the lingula of the left upper may be just as incapacitating as the original disease itself.

Pre-operative Preparation

There seems little doubt but that the use of the antibiotic penicillin pre-operatively has contributed to a safer, smoother postoperative course. Its use after operation is likewise important. Penicillin aerosol and/or penicillin intramuscularly effectively decreases the amount of expectoration and liquefies the material, thus permitting easier and more effective expectoration. These procedures, together with postural drainage, allow the patient to go through operation with a decreased hazard of postoperative atelectasis or pneumonitis from dissemination of the bronchial secretions.

Position During Operation

The contralateral spill-over of secretions during operation has been recognized as a distinct hazard when the patient is operated upon in the side position. To obviate this risk the patient is placed in the face-down position. This new operative positioning has been employed for all types of intrathoracic surgery for the past three years.[6] With the patient suspended by the pelvis, shoulders, and face, the head can be kept lower than the remainder of the body, thus causing secretions to flow toward the patient's mouth without the tendency to contralateral contamination that occurs with the patient in the side position. This face-down position, although developed originally for resection in tuberculous cases, has been very beneficial for resection in cases of bronchiectasis because of the frequently large volume of bronchial secretion.

Patients in the prone position appear to withstand an open pneumothorax better than when lying on the side as the good lung is not encumbered by the weight of the heart and mediastinum, and there appears to be less tendency to mediastinal shift with respiration.

We also believe that the use of procaine anesthesia with endotracheal intubation under cocaine topical anesthesia, rather than the use of a general anesthetic, promotes a smoother convalescence and lessens postoperative pulmonary complications. The cough reflex is not completely inhibited during operation and becomes active immediately afterwards for the patient is conscious enough to cooperate.

Technique of Segmental Resection

Each bronchopulmonary segment has its own bronchus, arterial blood supply, and venous return. Bronchus and artery penetrate through the hilum into the substance of the segments. The venous channels return along the surface of the segments. It is possible to apply the principles of individual ligation technique, as used in lobectomy and pneumonectomy, to the bronchopulmonary segments. After the artery, bronchus, and vein to a segment have been ligated and divided, it is then possible by gentle blunt dissection to separate the diseased from the normal segments. Before this separation is started, the normal segments are inflated by raising the intrabronchial pressure. The line of demarcation then stands out clearly and facilitates separation. The plane is comparatively avascular, and the actual splitting of the lobe is done by blunt dissection mainly by the thumb and forefinger. After the bronchus, artery, and vein to the segment have been divided, the only structure holding the segments together that needs to be separated by sharp dissection is the visceral pleura. This may be divided before the actual splitting of the lobe or afterward. We prefer to do this step last as it is occasionally difficult to accurately define the intersegmental pleural line until the segments are separated. The surface of the remaining segment may bubble slightly, but this usually stops after a moist cottonoid pad has been applied for a few moments. The use of clamps on the lung tissue is avoided and considered hazardous. It is impossible to place the clamps precisely in the intersegmental plane. Either too little or too much tissue will then be excised. No attempt is made to suture over the edges of the remaining segment as it has been found to be not only unnecessary but unwise. Suturing traumatizes the normal remaining tissue and reduces its volume.

Postoperative Care

The after care of the patient following segmental resection is similar to that following a routine lobectomy. Attention is directed toward the maintenance of a free airway and re-expansion of the remaining pulmonary tissue. As a rule two intrapleural catheters are left in the chest—one posteriorly and one anteriorly—and both are connected to under-water drainage with moderate negative pressure of 4 to 8 centimeters of water. The drainage tubes are usually withdrawn 48 to 72 hours after operation provided there is roentgenographic evidence that the pleural space has become obliterated and the lung expanded. In some instances it is necessary for the tube to remain longer if there is still evidence of leakage of air from the pulmonary surface.

Oxygen is not ordinarily required postoperatively. It is felt that

in the absence of definite indication for its use, the deeper breathing that takes place in a normal atmosphere is advantageous in that it encourages more rapid re-expansion. Patients are urged to cough and raise voluntarily, and their position is changed frequently. Any evidence of retained secretions or atelectasis calls for prompt transnasal tracheo-bronchial aspiration or the more positive removal by bronchoscopy. An uneventful postoperative course is largely dependent on the maintenance of a free airway and prompt, complete pulmonary expansion.

Results

The technique of segmental resection has been applied to all segments of all lobes for various conditions. This report, however, is limited to patients with bronchiectasis in whom segments of two or more lobes, or a lobe and one or more segments, were so removed. In the beginning segmental resection was employed only in patients with multi-segmental and bilateral disease. For the patient with unilobar involvement, lobectomy may still be preferable as conservation of all healthy pulmonary tissue is not as imperative.

In our series of patients with bronchiectasis, a total of 53 cases have been treated by one or more segmental resections. Inasmuch as we believe at present the procedure is primarily indicated in multilobar disease, this report has been confined to the 39 patients that have had multilobar resection.

Figure 5 shows the location of the various segments removed. We have found it usually more practical to remove all the basal segments of the lower lobes (3 on the left side and 4 on the right) as a single unit. It is possible to remove individual basal segments, but in bronchiectasis all three left and four right basal segments are commonly involved and therefore the basal group will usually require their group removal. In one bilateral case the individual posterior basal segment was removed and the other three healthy basal segments were preserved.

The discrepancy between Figures 1 and 2 in the number of patients and the number of operations is due to two factors. First, many of these patients have bilateral processes requiring bilateral operations. Often a period of 3 to 6 months or more may elapse between stages. Twelve patients with bilateral disease have not yet had the contralateral lesions excised, but for completeness all the cases have been included. Second, it is our practice to do the most involved side first. Some of these patients are so greatly improved that the minimal symptoms from the contralateral side have not as yet warranted the second operation.

The preponderance of resections of the basal segments of the

left lower lobe and the lingular segments of the left upper lobe is due to this combination being most frequent in our multilobar group, and to our practice of generally operating on the left side first when the lesion is about equally distributed between the two sides. As mentioned before, the uncompleted bilateral cases thus weight the figures in this group.

Fourteen patients have had bilateral operations completed. As a group they are strikingly better than the patients with bilateral disease treated by bi or trilobar resection. The pulmonary reserve of most is practically as good as before operation as no normal-functioning pulmonary parenchyma has been sacrificed.

There was one fatal case in the 39. This one has been reported elsewhere. The death was not related to the type of resection performed.

Morbidity and hospitalization for the segmental cases is lengthened on the average by 4 to 7 days. This is due in part to an occasional persistent air leak from the pulmonary surface and also to a desire to keep these patients under observation a little longer than the patients following a routine lobectomy. We believe the increased morbidity is not significant when the final result is so much superior. When the morbidity has been reduced

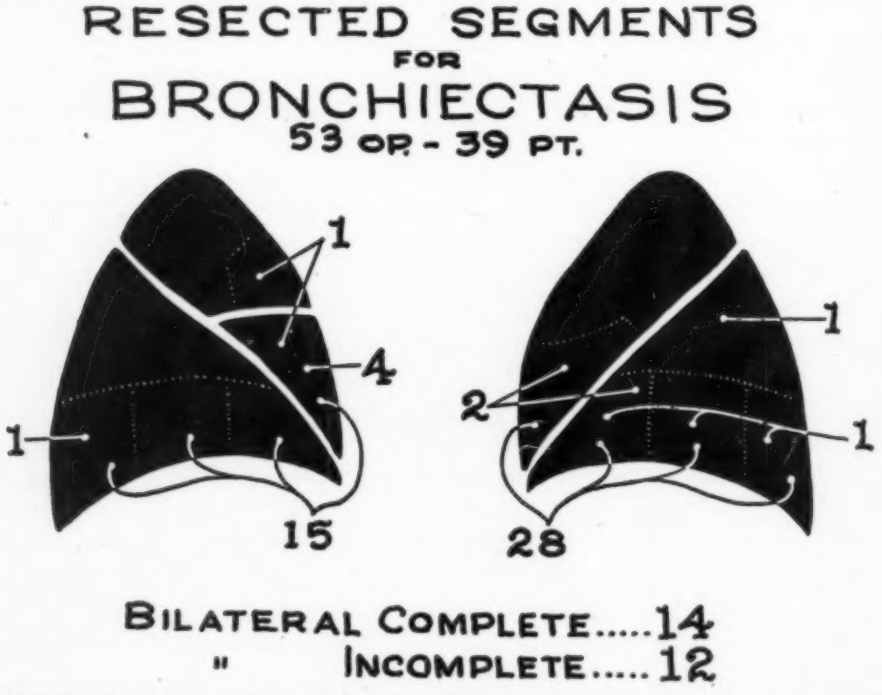


FIGURE 5: Diagram illustrating the various segments that have been removed for bronchiectasis. There were 39 patients, 26 of whom had bilateral disease. Upon these patients 53 operations have been completed.

to that of simple lobectomy, then segmental resection should be extended to include the patient with unilobar disease.

SUMMARY

Bronchiectasis is usually a segmental rather than a lobar disease process. Surgical therapy should, ideally, remove all the involved segments without sacrificing any normal pulmonary tissue. Refinements and advances in diagnosis, localization, and surgical therapy now make such a goal attainable.

Fifty-three operations in 39 patients, with one death, are reported. All had at least two segments, or a lobe and a segment, excised. There are 26 cases with bilateral bronchiectasis in which bilateral operations have been completed on 14.

Segmental resection is especially applicable to patients with multilobar involvement where conservation of all normal pulmonary tissue is essential.

RESUMEN

Por lo general la bronquiectasia es un proceso morboso segmentario más bien que lobular. Lo ideal es que la terapia quirúrgica extirpe todos los segmentos invadidos, sin sacrificar ningún tejido pulmonar normal. Los refinamientos y adelantos en el diagnóstico, la localización y la terapia quirúrgica permiten ahora que se pueda alcanzar este objeto.

Se informa sobre 53 operaciones en 39 enfermos, uno de los cuales murió. En todos ellos se extirparon por lo menos dos segmentos, o un lóbulo y un segmento. Hay 26 casos con bronquiectasia bilateral, en 14 de los cuales se han completado operaciones bilaterales.

La resección segmentaria es aplicable especialmente a enfermos con afección multilobular, en los que es esencial que se conserve todo el tejido pulmonar normal.

REFERENCES

1 Blades, Brian: "Conservation of Lung Tissue by Partial Lobectomy," *Ann. Surg.*, 118:353-365, 1943.

2 Bradshaw, Howard H., Putney, Floyd J., and Clerf, Louis H.: "The Fate of Patients with Untreated Bronchiectasis," *J.A.M.A.*, 116:2561, 1941.

3 Churchill, E. D., and Belsey, Ronald: "Segmental Pneumonectomy in Bronchiectasis. The Lingula Segment of the Left Upper Lobe," *Ann. Surg.*, 109:481-499, 1939.

4 Clagett, O. T., and Deterling, R. A. Jr.: "A Technique for Segmental Pulmonary Resection with Particular Reference to Lingulectomy," *J. Thor. Surg.*, 15:227-237, 1946.

5 Jackson, C. L., and Huber, J. F.: "Correlated Applied Anatomy of the Bronchial Tree and Lungs with a System of Nomenclature," *Dis. of Chest*, 9:1, 1943.

6 Overholt, Richard H., and Langer, Lazaro: "A New Technique for Pulmonary Segmental Resection," *Surg., Gynec., Obstet.*, 84:257-268, 1947.

7 Perry, Kenneth M. A., and King, Donald S.: "Bronchiectasis," *American Rev. Tuberculosis*, 41:531, 1940.

Discussion

FRANK P. COLEMAN, M.D., F.C.C.P.
Richmond, Virginia

In planning the resection of a portion of a lobe or lung for bronchiectasis, the importance of accurate localization of the disease cannot be over-emphasized. The establishment of a diagnosis by the routine roentgenogram is neither reliable nor indicative of the extent of the disease. Bronchoscopy aids in excluding secondary bronchiectasis, but the segmental distribution of primary bronchial dilatation cannot be depicted with any degree of accuracy by this method. The absolute diagnosis and segmental distribution of the disease is dependent upon good lipiodol filling of all the bronchi to the multiple segments in both lungs. Many failures in the surgical treatment of this disease can be attributed to lack of pre-operative delineation of all the involved segments.

The characteristic broncho-pulmonary segmental distribution of bronchiectasis has been emphasized today. In a series of 75 bronchiectatic patients subjected to resection, I found the disease to be unilateral in 54 and bilateral in 21 cases. Of 34 cases with a single lobe involved, 4 had a single diseased segment and the remaining showed multiple diseased segments. Forty-one patients revealed pre-operative involvement of more than one lobe. In this series of cases the extent of the resection did not always parallel the extent of the disease. Marked symptomatic improvement following resection of the most advanced disease confined to one lung caused a few of the patients to decline an operation on the contralateral lung.

Doctors Overholt, Betts and Woods' observations and technical contributions to this subject are timely, emphasizing the importance of preserving healthy functional lung tissue and of offering a chance of a cure to many bronchiectatic patients who have heretofore been denied surgery because of extensive multilobar disease. It would seem that re-evaluation of many of the so-called hopelessly advanced cases of bronchiectasis is in order, for segmental resection of multiple lobes is being executed with safety terminating in a cure.

The individual treatment of segmental hilar structures in performance of a partial lobectomy requires a detailed and thorough knowledge of anatomy. Although excellent and detailed representation of the usual anatomy of some of the segments were presented and previously published by the authors, I feel as if the surgeon should devote considerable time to dissecting fresh autopsy specimens prior to carrying out the operation on a patient. De-

lineation of the various pulmonary segments by inflation and deflation of the fresh autopsy specimen is not too clearly defined on the surface; however, in the living the segmental surface demarcation of the diseased segment is usually obvious after ligating the bronchovascular structures to the segment. Segmental resection by the individual ligation technique is applicable to all segments of the lungs; however, removal of the basal segments (as a unit), lingula, superior segments and anterior segments (upper lobes) are more accessible and easier in execution. Excluding the lingula segment, I have performed 16 segmental resections for bronchiectasis and 4 such resections for other pathology. The surgical approach, identification and treatment of the bronchovascular anatomical structures, and surface delineation of the segment have been the same as presented by Doctors Overholt, Betts and Woods. The intersegmental plane is developed by blunt dissection by the authors whereas we have used sharp dissection cutting boldly through the lung parenchyma in a line of the intersegmental plane. Bleeding is negligible in that the parent artery to the respective lobe is temporarily occluded by drawing taut a previously placed ligature of umbilical tape. The raw surface of the remaining segment is covered by a pleural graft. In this small group of cases there were no postoperative empyemas; however, the evacuation of bronchial secretions is not as effective as in the average postoperative lobectomy. Postoperative bronchoscopy revealed in two cases where the remaining superior segment of the lower lobe was not aerating well anterior rotation of the segment orifice. This may be a factor in the development of postoperative atelectasis of this particular segment.

Noxious Gases and Bronchiectasis*

DUANE CARR, M.D., F.C.C.P., W. E. DENMAN, M.D., and E. F. SKINNER, M.D.

Memphis, Tennessee

During the past three years we have had the opportunity of examining a large number of patients who have been employed in a government arsenal where shells are loaded with mustard gas. While working in the shell loading department, these patients are subjected to variable amounts of mustard gas in the atmosphere. The histories of these patients follow a fairly uniform pattern, as do the findings on physical examination, roentgenological examination with bronchograms and at bronchoscopy. We have seen these patients in all stages of development of the disease found in the bronchial tree, except the initial or acute stage, which is described for us by the Staff of the Station Hospital at the arsenal. Observation of these patients has given us a fuller understanding of the pathological changes which occur as a result of chemical burns of the bronchial mucous membrane, not only from mustard gas but from other chemical agents with which patients come in contact in civilian life.

The history of the acute phase of the chemical burn usually states that after a period of repeated exposure ranging anywhere from three weeks to six or seven months the patient begins to show signs of irritation of the conjunctival and respiratory mucous membranes. The patients develop conjunctivitis, photophobia, lacrimation, impaired vision, loss of taste and smell sensation, nose bleeds, sore throat, difficulty in swallowing, hoarseness, chest pain, retrosternal soreness, wheezing and dyspnea. In addition there may be anorexia, vomiting, weight loss, general weakness, insomnia, and irritability. When the patient is seen in the outpatient department of the Station Hospital, he is given symptomatic treatment and perhaps several days sick leave or temporary transfer to another department. After removal from the atmosphere containing mustard gas the symptoms all disappear except those referable to the respiratory tract.

The patients were referred to us by the United States Marine Hospital three months to two years following exposure. The acute symptoms had subsided but in their place has appeared a chronic cough of considerable severity, particularly prominent on retiring

*Presented at the Annual Meeting, Arizona Chapter, American College of Chest Physicians, Phoenix, Arizona, May 1, 1946.

at night or upon arising in the morning. The cough is usually productive of sputum varying from two tablespoonfuls of clear mucoid sputum to one or two cups of mucopurulent sputum daily. There is usually a period of two months or so in the history when the sputum was blood streaked. Occasionally frank hemoptysis has occurred. As the disease progresses the patients complain of dyspnea, excessive fatigue, wheezing and, not infrequently, night sweats. Pain in the chest is a prominent symptom and often a substernal soreness or feeling of rawness. Spontaneous remission appeared in the histories of only three patients. In all others the symptoms are of progressive severity. A number of the patients complain of loss of weight but this is not a uniform observation.

On physical examination, these patients are found to have a low grade fever usually not higher than 99.4° F. Those patients having the most purulent sputum due to a secondary infection of their bronchi usually show an elevation of pulse rate as well. The most constant physical findings are those of bilateral coarse moist rales with numerous wheezes heard on both inspiration and expiration throughout both lung fields. In several patients, distant voice and breath sounds are heard.

The laboratory of the United States Marine Hospital routinely obtained blood counts, sputum examinations for tubercle bacilli, urinalysis, blood sedimentation rate and blood tests. The red cell count and hemoglobin levels were usually within normal limits, indicating that the mustard gas itself had no particular affinity for the hematopoietic system. The white cell counts varied from normal to the upper limits of approximately 15,000 white blood cells. This appears to depend more upon the amount of secondary infection in the bronchi than on any other factor. The differential count was uniformly within normal limits. The blood sedimentation rate varied from normal to 20 mm. in the first hour, the large majority of determinations falling between 12 mm. and 17 mm. per hour. The few patients with sputum positive for tubercle bacilli were excluded from this series. Serological tests were within normal limits for the population group studied.

X-ray films of standard technique are usually within normal limits in appearance. Rarely, evidence of interstitial fibrosis is seen in the bases. Eleven of the patients show an advanced stage of pulmonary emphysema as demonstrated by the depression of the diaphragm with blunting of the costophrenic angles and unusual penetrability of the lung fields.

Bronchograms were made on one hundred forty-four of these patients and repeated at six month intervals on many of them. The earliest change noted in the bronchi is an irregularity of the mucous membrane, the outline of the bronchial walls appear-

ing roughened or ragged. This is not to be confused with the spotty filling obtained when a great deal of secretion is present in the bronchi. In our terminology this roughening indicates damage to the mucous membrane and the diagnosis of bronchitis is made. Unless roughening and irregularity is definitely present, the patients are listed as being "without demonstrable disease," regardless of the history. Of the one hundred forty-four patients upon whom one or more bronchographic studies were carried out, fourteen showed no demonstrable damage to the bronchi in the bronchograms. Sixty-four of the patients showed definitely ragged and irregular bronchial walls and were given a diagnosis of bronchitis.

The next phase of the disease demonstrated in the bronchograms is a dilatation of the terminal bronchi. These patients, as well as those with a marked dilatation of only two or three secondary bronchi, were given a diagnosis of minimal bronchiectasis. Forty-one of these patients exhibited just such changes.

Cylindrical dilatation of moderate extent in two or three lobes or even marked dilatation confined to one lobe is considered moderately advanced bronchiectasis in this series. Thirty of the one hundred forty-four patients examined received this designation.

Extensive cylindrical and saccular dilatation was found in sixteen patients who are termed far advanced bronchiectasis.

Of the one hundred forty-four patients, eighty-seven exhibited a bronchiectasis of some degree. We recognize that our designation of minimal bronchiectasis may be questioned by some authorities. However, there is a total of forty-four patients, almost one-third of the entire group, showing moderately advanced or far advanced bronchiectasis which is beyond question.

Bronchoscopic examination was carried out on eleven of these patients to learn more about the pathological changes in the bronchial mucous membrane in relation to the appearance of the bronchograms. The mucous membrane of the major bronchi is inflammed and edematous in those patients with the more active symptoms. In two of the patients who had previous bronchograms and who had been bronchoscoped, the bronchoscopy was described as normal. However, biopsy specimens taken from the bronchial wall showed evidence of a mucous membrane which had been destroyed and which had undergone regeneration. The epithelium varied in cellular structure from an almost squamous cell to a low columnar epithelium totally without cilia. Others showed no evidence of active inflammation but the bronchial wall appeared irregular, scarred and the usual velvety sheen of a normal ciliated epithelium was lacking.

Our observation of these patients leads us to postulate that the pathological changes begin with an acute chemical irritation or

burn of the bronchial mucous membrane followed by inflammation resulting in the loss of bronchial elasticity and ciliary action. This interferes with the normal evacuating mechanism of the lungs. Secondary infection supervenes, causing further localized damage to the bronchial wall and, in many instances, leading to sufficient erosion to destroy the elastic tissue fibers. In those areas bronchiectasis results. It appears to be during the period of ulceration and destruction that the blood streaked sputum and hemoptyses occur.

We have been inclined to apply the term hypertrophic bronchitis to those patients in whom wheezing, dyspnea and a sense of tightness in the chest are the outstanding symptoms and in whom the bronchograms show attenuated bronchi throughout. These are the patients with the inflammed and swollen mucous membrane. However, when the condition evolves into the stage in which regenerated but atrophic epithelium is found, the term atrophic bronchitis might well be more applicable. It is the group of patients with wheezing, attenuated bronchi, and hypertrophied or swollen mucous membrane which develop the severe pulmonary emphysema seen in eleven of these patients.

As consultants for the United States Marine Hospital we have not had the opportunity of personally supervising the treatment of these patients directly. Our recommended treatment has been a routine of fifteen grains of ammonium chloride four times a day, postural drainage with forced coughing for two or three minutes four times a day, a high fluid intake and complete abstinence from smoking. Removal from the irritating fumes is likewise advised. For acute febrile episodes, or for all patients whose purulent sputum is excessive in amount, we are now recommending that penicillin both parentally and by inhalation be given.

To date no lobectomy has been performed on any of these patients although a number have had demonstrable bronchiectasis in only one lobe. So many of the patients on repeated examinations with bronchograms have shown progressive disease that we have hesitated to perform a resection until the bronchiectasis has been demonstrated to be stable and to have reached its maximum development.

The bronchial drainage routine outlined, when faithfully carried out, results in a marked reduction in cough and sputum, improvement in appetite and usually in a marked gain in weight. In only two patients have we seen any remarkable reduction in the caliber of the dilated bronchi. Few of the patients admit relief from dyspnea on heavy exertion. We find no record of recurrence of bloodstreaked sputum or hemoptysis in a patient following the bronchial drainage routine faithfully. Those patients whose mental

capacity permits them to do work other than heavy manual labor find it possible to hold a job as long as the bronchial drainage routine is carried out.

SUMMARY

The effects of repeated exposures to varying concentrations of mustard gas as reflected in the bronchial tree are described, based upon observation of one hundred forty-four patients who were employed in a gas shell loading plant. It may reasonably be assumed that similar changes in the bronchi will be observed as a result of chemical burns due to other agents such as concentrated ammonia fumes, slack lime, etc., as encountered in industrial work.

It is shown that forty-six of one hundred forty-four patients have developed a definite moderately or far advanced bronchiectasis. An additional forty-one patients exhibit a minimal bronchiectasis. In combination with minimal bronchiectasis, or as the only demonstrable anatomical change, bronchitis was demonstrated in sixty-four patients. Asthmatic bronchitis associated with hypertrophic bronchitis or atrophic bronchitis was found in forty patients. Eleven patients had advanced bilateral pulmonary emphysema.

The treatment applied to all cases consisted of a bronchial drainage routine including ammonium chloride, 15 grains four times a day, postural drainage with forced coughing for two or three minutes four times a day, a high fluid intake and abstinence from smoking. Removal from contact with mustard gas was likewise recommended. Patients with asthmatic symptoms were benefited by supplemental use of Tedral (theophylline, ephedrine and phenobarbital) as a "bronchial relaxing" agent. Penicillin therapy by injection and by inhalation has been more recently used by those patients with marked secondary infection in the bronchial tree.

The majority of patients faithfully following this routine have been able to work at sedentary occupations which do not require heavy physical exertion. The emphysema patients are totally disabled.

As yet no pulmonary resection has been carried out in this group because of the progressive character of the pathology observed.

A review of this group of patients five years from now will be of extreme interest and help in evaluating disability following exposure to noxious fumes and chemical agents.

RESUMEN

A base de observaciones llevadas a cabo en ciento cuarenta y cuatro pacientes que trabajaron en una planta en la que llenaban granadas con gas, se han descrito los efectos sobre el árbol bron-

quial de repetidas exposiciones a diferentes concentraciones del gas mostaza. Es razonable suponer que se observarán alteraciones semejantes en los bronquios como resultado de quemaduras químicas causadas por otros agentes, tales como vapores de hidróxido de amonio concentrado, hidróxido de calcio, etc., que se usan en el trabajo industrial.

Se demuestra que en cuarenta y seis de los ciento cuarenta y cuatro pacientes se ha desarrollado una bronquiectasia bien definida, desde moderada a muy avanzada. Otros cuarenta y un pacientes manifiestan una bronquiectasia mínima. Se comprobó bronquitis en sesenta y cuatro pacientes, ya en combinación con bronquiectasia mínima o como el único cambio anatómico demostrable. En cuarenta pacientes se encontró bronquitis asmática asociada con bronquitis hipertrófica o atrófica. Once pacientes tenían enfisema pulmonar bilateral avanzado.

El tratamiento aplicado a todos los casos consistió de un programa rutinario de canalización bronquial que incluyó la administración de cloruro de amonio (un gramo cuatro veces al día), canalización por postura con tos forzada por dos o tres minutos, cuatro veces al día, la bebida de grandes cantidades de líquidos y abstinencia de fumar. También se recomendó cesar el contacto con el gas mostaza. Los pacientes con síntomas asmáticos obtuvieron efectos beneficiosos mediante el uso suplementario de Tedral (teofilina, efedrina y fenobarbital) como agente "dilatador de los bronquios." Más recientemente, el tratamiento con penicilina por inyección y por inhalación ha sido empleado en esos pacientes con marcada infección secundaria del árbol bronquial.

La mayoría de los pacientes que han observado fielmente este programa rutinario han podido trabajar en ocupaciones sedentarias que no requieren esfuerzo físico pesado. Los pacientes con enfisema están totalmente incapacitados.

Hasta ahora no se ha llevado a cabo ninguna resección pulmonar en este grupo debido al carácter progresivo de los procesos patológicos observados.

Un análisis de este grupo dentro de cinco años sería muy interesante y ayudaría a apreciar la incapacidad causada por la exposición a vapores y agentes químicos nocivos.

REFERENCES

1 Morgenstern, Capt. Philip, M.C., A.U.S., Koss, Capt. Frank R., M.C., A.U.S., and Alexander, Wm. W.: "Residual Mustard Gas Bronchitis," To be published.
2 Henderson, Y., and Haggard, H. W.: "Noxious Gases and the Principles of Respiration Influencing their Action," *Reinhold Publishing Corporation*, New York, New York, 1943.
3 Gilchrist, H. L., and Matz, P. B.: "The Residual Effects of Warfare Gases," *U. S. Government Printing Office*, Washington, D. C., 1933.

Work Capacity: Its Role in the Treatment of Tuberculosis*

BENJAMIN L. BROCK, M.D., F.C.C.P.[1] and
BERNARD D. DAITZ, M.S.P.H.[2]
Oteen, North Carolina

The treatment of tuberculosis, based as it is upon the concept of bed rest, has as its ultimate objective the restoration of the patient to a state of relative well-being and a capacity for participating in a program of work which is consistent with his handicap. For the physician who treats the tuberculous patient, for the others who are interested in the other aspects of his rehabilitation, and for the patient himself, the problem of absolute bed rest has been a vexing one. Countless papers have been written dealing with the factors which prompt patients to leave tuberculosis hospitals against medical advice, and not a few have referred to the inability of patients to adjust to the bed rest regimen.

The physician must rely for the most part upon his experience and empirical judgment to determine at what time the patient may begin his activity and how much activity he may undertake. Neither qualitative nor quantitative yardsticks have yet been developed by which the amount of activity in which a tuberculosis patient participates may be measured, with perhaps the possible exception of the time factor. Thus a patient for whom activity is prescribed is told that he may devote a half hour twice each day to reading, studying, occupational therapy, or related work.

With the development of rehabilitation programs for tuberculous patients during recent years, it has become increasingly apparent that evaluation of their work capacity constitutes a significant factor in assisting them to evolve vocational readjustment plans consistent with their physical limitations. The ability of the patient to sustain, without untoward effects, an activity program when his pulmonary lesion has become stabilized enters into the evaluation of his clinical status. Thus the patient

---

*Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors. Presented at the 4th Annual Meeting, Southern Chapter, American College of Chest Physicians, Miami, Florida, Nov. 4, 1946.

1 Clinical Director, Veterans Administration Hospital, Oteen, N. C.

2 Executive Director, Medical Rehabilitation Service, Veterans Administration Hospital, Oteen, N. C. Present Address: Director, Tuberculosis Rehabilitation, Medical Service, Veterans Administration, Washington 25, D. C.

with an unstable pulmonary lesion is classified as "active" clinically or pathologically, and is generally kept at absolute bed rest until the lesion stabilizes. Activity, in most instances, is permitted only after a lesion has become stable. The patient then progresses through graduated levels of activity, more or less following a "time table," unless a flare-up occurs. At the time of discharge, the patient who has reached the classification "arrested" has a work capacity for one hour of walking exercise twice daily, or its equivalent, having shown that he has been able to sustain such a program for a period of two months without reactivation of his symptoms of his lesion. For some time now, it has been obvious to many of us that this activity level is insufficient to meet the conditions which the patient must encounter in the work-a-day world. Furthermore, the physician is unable to predict with any assurance just how well the patient will tolerate a given work experience because in most instances it has not been possible in the hospital to observe the patient's reaction to activity similar to that which he will be subjected to in the job of his choice.

Recent studies have yielded information which may contribute to a better understanding of the physiology of bed rest and exercise and may possibly suggest useful techniques which may be applied in the management of pulmonary tuberculosis. In a series of carefully controlled observations, using conscientious objectors, Keys[1] found that, following a six week period of absolute bed rest, otherwise normal subjects demonstrated significant signs of physical and mental deconditioning. He reports that, while there is relatively little loss in simple muscular coordination; endurance, postural coordination, and adjustment are quickly impaired. Cardiocirculatory capacity and efficiency are reduced, the blood volume diminishes, the heart becomes smaller, and a relative tachycardia develops even in basal rest. He also has found rather clear indications that tissue metabolism is affected. The body goes into negative nitrogen balance, requiring an appreciable increase in protein intake to restore normal balance. A negative calcium and potassium balance is also observed, further suggesting that tissue disintegration is taking place. Thiamine, vitamin C, and riboflavin appear in the urine in unusual amounts. These observations are especially interesting since similar phenomena are not uncommonly associated with the clinical findings in tuberculosis. Barr and his co-workers[2] have reported substantially similar data and, in addition, a marked hyper-reaction to the tilt table. These findings become even more significant when related to the observations of Bray[3] who questions the value of absolute bed rest in treating pulmonary tuberculosis in patients who show good nutrition and are symptom free or practically so. Bray cites the paucity

of published studies which contain strong evidence that strict bed rest is significantly more effective than prescribed and graduated activity. He further observes that curtailment of activity has a definitely deleterious effect upon muscle tone and mental attitude.

Recognition of the fact that patients, subjected to strictly limited opportunities for exercise or related activity during their illness, developed tangible evidence of both physical and mental degeneration, prompted the development by the Army and the Navy of reconditioning programs in the various service hospitals during the recent World War. This type of therapy materially helped to restore thousands of men to maximum levels of efficiency and, simultaneously, shortened the length of their hospital treatment. Patients were brought to a peak of physical fitness through a reconditioning process which included physical therapy, occupational therapy together with competitive team play, supervised and graduated calisthenics, and active recreation. The mental aspect was not neglected. Patients were encouraged to enroll in a variety of study courses which were developed by the United States Armed Forces Institute. Many patients in Army and Navy hospitals were thereby able, successfully, to complete and receive academic credit for the educational work which they took. The rehabilitation value of this reconditioning program has been adequately demonstrated.

These observations suggest that basal physiologic rest should be prescribed with as much care as is an activity regimen. It should be noted that none of the studies which have been cited discounted the value of bed rest as a therapeutic measure. If any conclusion may be drawn, it would suggest that greater consideration be given in prescribing basal physiologic rest, to the amount required and for what length of time. Furthermore, the effects of too much rest should be considered. This concept does not seem to be inconsistent with the present principles of bed rest therapy in the treatment of pulmonary tuberculosis.

It therefore seems quite clear that to be effective, a work capacity program for tuberculosis patients should be predicated upon a consideration of the following: the factors which determine how soon a patient may be permitted to begin an activity program; the factors which determine the amount and type of activity prescribed; and the factors which determine when and by how much, activity may be increased.

There is no doubt regarding the necessity for restricting the activity of the patient who is toxic and shows the characteristic elevated temperature, pulse, and erythrocyte sedimentation rate, is in poor nutrition, or whose pulmonary lesions are in any way

suggestive of an acute tuberculous process. At such time, however, as the toxicity has subsided, the patient has improved nutritionally, and there is evidence that the pulmonary lesion is retrogressive, it would appear that an activity prescription may be considered.

Based upon the studies which have been cited and some work done by Covalt and Covalt,[4] we are presently studying a program of mild, graduated exercises which are to be given by physical therapists under medical supervision. These exercises are particularly designed to teach the patient how to relax and thereby enable him to tolerate a bed rest regimen which will still constitute the major portion of his therapy. At the same time, however, it is intended that these exercises will initiate the restoration and maintenance of muscle tone. An indication of the type of exercise which will be employed may be obtained from a brief description of the first series which will be prescribed for the patient who has been on complete bed rest and has reached the point of being able to sustain a minimum of activity. The exercises are demonstrated to the patient by the physical therapist to insure that he understands the procedures.

(The following exercises are done by the patient while lying flat on his back. They are repeated four times each day at prescribed times):

1. Curl the toes downward and at the same time bend the foot upward at the ankle. Relax and repeat three times. Exercise one foot at a time.

2. Repeat exercise number 1, but this time rotate the foot upward and inward so that the sole of the foot may be seen. Relax and repeat three times.

3. Keep the leg straight. Tighten the muscle on the upper surface of the thigh, pulling the kneecap upwards. Relax and repeat three times.

4. With the legs flat on the bed and about twelve inches apart, rotate the legs inward keeping the knees straight. Relax and repeat three times.

5. Clench the fingers of your hand to make a fist. Relax, stretching the fingers open and apart. Relax and repeat three times. Do this exercise one hand at a time.

The patient's reaction to this exercise program will be closely observed. If it is found that he is able to sustain the preliminary series, the second set of exercises will be prescribed and his reaction similarly noted. In this manner, we propose to increase the amount of activity and simultaneously the length of time. Integrated with this exercise program and depending upon the patient's interests, he will be permitted to read and sit up for

his meals. The total amount of such activity will be kept within the time limit specified by the physician. As the patient demonstrates continued progress in dealing with his disease, the activity prescription will be increased and occupational therapy and educational therapy facilities will be introduced. These will be determined by evaluating the patient's interests and aptitudes.

It should be evident that the only criteria which we now have are those which indicate that the patient shows no untoward elevation of temperature, pulse rate, or sedimentation rate of the red blood cells; and, further, that roentgenologically his lesion shows continued retrogression. However, as patients progress to ambulatory status and their activity program becomes diversified, the problem of evaluation becomes more complicated. The physical demands of different types of activity vary. Consequently, walking along level ground does not require as much energy output as climbing a hill. Similarly walking may logically be expected to require more effort than work at a desk or a work bench. The effect of these factors on fatigue must also be considered. The determination of activity equivalence is therefore a problem which will require special study. We propose to study our patients as they perform at their various activities and record the effects of measured amounts of work upon body temperature, pulse rate, erythrocyte sedimentation rate and similar physiologic reactions. It may be particularly interesting to observe the effect which such a program of graduated exercise and work will have upon calcium and potassium balance, and also upon the thiamine, vitamin C and riboflavin output. It would also be of particular interest to make these determinations in individuals with active pulmonary tuberculosis who have not as yet begun their bed rest regimen. Because our program is just now being developed, we have not been able to obtain data for inclusion in this paper. We are hopeful that this will be possible in the near future and that this discussion will stimulate investigation of this question by others. It seems possible that such study may yield some information which may serve as the basis for a better understanding of the problem of bed rest and work capacity.

While seemingly unrelated to the question of work capacity, the deformity which not uncommonly follows thoracoplasty does have associated with it limitation of arm and shoulder function and, of course, the mental reaction of the patient. It may consequently be of interest to report on the program which is being developed at the Veterans Administration Hospital at Oteen, which we expect will prevent the deformities and other functional defects which have been referred to. The success of similar programs which were employed by British workers and later in our Army

hospitals has recommended this approach to us. By individual preoperative and postoperative treatment and instruction given the patient by the physical therapist under the supervision of the surgeon and the physician-in-charge of Medical Rehabilitation, the patient is taught the principles of correct posture. Following surgery a series of mild, graduated exercises is prescribed and the patient is encouraged to use the arm on the operated side rather than keep it splinted to his side and inactive.

The work capacity program which we are developing is the principal mission of the Medical Rehabilitation Service, following the pattern so successfully demonstrated by the convalescent programs developed by the Armed Forces. As established by the Veterans Administration, the Medical Rehabilitation Service at our hospital at Oteen, North Carolina, which is almost wholly devoted to the treatment of tuberculosis, consists of four component units: Physical Therapy, Occupational Therapy, Educational Therapy, and Manual Arts Therapy. Under medical supervision, the work of each is coordinated to provide the patients diversified yet regulated activity which is both therapeutic and purposeful. As early as is consistent with his physical condition and his intellectual interests, each patient is permitted to participate in work, study and recreation designed both to enable him to explore his interests and aptitudes in a variety of vocational pursuits and also to enable the patient's physician to observe his ability to sustain such a program of activity.

SUMMARY

The concept of work capacity as applied in the treatment of pulmonary tuberculosis has been reviewed in the light of several recent studies of deconditioning phenomena. The question has been raised as to whether prescribed and carefully graded exercises may be effective in minimizing physical deconditioning of tuberculous patients if employed prior to ambulation. The need for more objective criteria for evaluating work capacity is suggested and a work capacity program outlined.

It is the hope of the authors that the views presented here will stimulate further study of these problems.

RESUMEN

A la luz de varios estudios recientes sobre los fenómenos del reacondicionamiento se ha analizado el concepto de la tolerancia al trabajo, en su aplicación al tratamiento de la tuberculosis pulmonar. Se ha discutido el problema de si los ejercicios prescritos y cuidadosamente graduados podrían ser eficaces para reducir al mínimo el reacondicionamiento físico de pacientes tuberculosos,

si se practicaran antes del período ambulante. Se indica la necesidad de emplear criterios más objetivos para avaluar la capacidad para el trabajo, y se bosqueja un plan de tolerancia al trabajo.

Los autores abrigan la esperanza de que las opiniones aquí presentadas sirvan de estímulo para que se emprendan estudios adicionales sobre estos problemas.

REFERENCES

1 Keys, A.: "The Physiology of Exercise in Relation to Physical Medicine," *Arch. Physical Med.*, 10:633-638, (Oct.) 1945.

2 Barr, D. P.: "Report to the Graduate Fortnightly of the New York Academy of Medicine," October 1945.

3 Bray, H. A.: "Strict Bed Rest in Pulmonary Tuberculosis," *Amer. Rev. Tuberc.*, 52:483-489, 1945.

4 Covalt, D. A., and Covalt, N. K.: "You Can Get Well Faster," *Brochure for Patients*, Veterans Administration, Washington 25, D. C.

Transitory Pulmonary Infiltration
(Loeffler's Syndrome)*

THEODORE L. SQUIER, M.D.
Milwaukee, Wisconsin

In 1932, Loeffler[1] described a series of five cases in which pulmonary shadows appeared and disappeared rapidly. Extensive lung changes were accompanied by minimal clinical symptoms and by high grade blood eosinophilia. In 1936, he[2] reported additional cases bringing the total number observed by him to 51. The symptom complex was recognized by its three chief features: (1) a roentgen shadow in the lung fields; (2) the transitory or migratory character of the infiltrations; and (3) the accompanying blood eosinophilia which ranged from 6 to 66 per cent. Fourteen of his 51 cases were discovered on routine fluoroscopic examinations of patients who had no symptoms directly referable to the chest. This surprising absence of constitutional disturbance was regarded by Loeffler as a fourth, and not unessential diagnostic feature. Acoustic signs usually were slight or might be completely lacking. In many cases, there was an irritative cough, often of considerable intensity, with or without stabbing chest pain. Expectoration was lacking or scant, yellowish, muco-albuminous, but poor in cells. Occasionally, but not always, a few eosinophile cells were found. When sputum was obtained, it was always negative for tubercle bacilli. In rare instances, he observed pleural participation in the process, and still more rarely, a very small circumscribed pleural effusion, distinguishable in the roentgenogram or on fluoroscopy from the infiltrations. These pleural phenomena were likewise fleeting in character. The infiltrations observed were compared by Loeffler to the transitory lesions of erythema nodosum. In both conditions, fleeting inflammatory reactions occur, and he believed them to be the result of an allergic tissue reaction to many different antigens.

Following Loeffler's description, a number of similar reports have appeared in the literature and the cumulative evidence has made it increasingly clear that the syndrome results from an allergic tissue reaction as was hypothesized by him. As was predicted, a great variety of noxious agents such as ascaris,[3] trichinal,[4] and amoebic infestation,[5,6] brucellosis,[7] the pollen of privet,[8] prontosil,[9] and gold[10] have been reported as causative agents.

*From the Department of Medicine, Marquette University School of Medicine, Milwaukee, Wisconsin. Presented at the Annual Meeting, Wisconsin Chapter, American College of Chest Physicians, Oct. 6, 1946.

Because the condition is essentially benign, the histological characteristics remained conjectural until 1942 when H. von Meyenburg[11] described eosinophilic pulmonary infiltrations found in four patients after accidental death. These infiltrations varied in form and shape, and they varied likewise in location. High grade eosinophilia in the exudative-inflammatory lung nodule was common to all. Eosinophilic bronchitis and bronchiolitis was present in two cases, but was lacking in the other two. Based on the lesions found, von Meyenburg expressed his belief that such infiltrations were not necessarily always transitory. Harkavy[12] reported a series of asthmatic patients in whom polyvalent sensitization was present. Symptoms were precipitated synergistically in at least seven of these patients. In one patient, subcutaneous injection of an autogenous vaccine composed of various bacteria grown from sinus washings repeatedly was followed by asthma, pulmonary infiltrations and petechiae which were regarded as indicative of an hyperergic vascular response. He emphasized that symptoms in bacterial allergy may be reactivated not only by the specific bacteria responsible for the initial sensitization, but also by heterologous bacteria, nonbacterial antigens and possibly by viruses. Such heterologous reactivation of symptoms is comparable to the Sanarelli-Schwartzman phenomenon. Patients with multiple shock tissues reacted not only in the bronchi, but in vessels of the pulmonary parenchyma and other tissues as well and characteristic transitory pulmonary infiltrations of the Loeffler type were observed repeatedly. The milder vascular reactions were regarded as reversible, and in eight patients of the group reported by Harkavy, removal of offending agents was followed by arrest and reversal of the disease process. However, repeated attacks resulted in chronic allergic inflammatory changes in the interalveolar septa and progressive vascular changes.

Allergic infiltrations are not confined to pulmonary tissues alone. von Meyenburg described eosinophilic infiltration of an epididymis removed surgically from a patient in whom transitory pulmonary infiltrations had been observed previously. Likewise, brawny infiltrations were observed by Harkavy in the thigh of a patient who had fleeting pulmonary infiltrations accompanying asthmatic attacks. Biopsy showed these lesions to be eosinophilic infiltrations. Klopstock and Steinitz[13] reported similar migratory reddened swellings which recurred in scattered areas of the body and were accompanied by increased leucocyte counts varying from 12,000 to 15,000 and high grade eosinophilia.

Marked perivascular collections of leucocytes were observed in the nodular infiltrates in two of von Meyenburg's cases. Harkavy emphasized the periarterial lesions in his fatal cases. Periarterial

lesions in association with transitory pulmonary infiltrations also were observed by Lindberg and Baggenstoss[14] in a 59 year old woman whose death from asthma occurred seven years after her first attack. For several months transitory pulmonary shadows of varying size had been accompanied by eosinophilia as high as 35 per cent. Necrotizing arteritis and arteriolitis were found at necropsy.

Three cases of transitory and migratory pulmonary infiltration which fulfill the criteria established by Loeffler are presented. In each instance there is a background of extrinsic asthma. The first two patients exemplify the early, completely reversible stage generally regarded as characteristic of Loeffler's syndrome. The third patient demonstrates a later stage in which roentgenograms now indicate increasing residual fibrosis of pulmonary tissues.

CASE REPORTS

Case 1: W.A.M., Male, age 27. Since his first year there have been recurrent attacks of asthma some of which were caused by contact with danders and dust. Symptoms were somewhat more severe during summer months, although attacks occurred throughout the entire year. Nasal polyps were removed at age twelve. At seventeen, he was given injections of dust, grass, and ragweed antigen for three years without appreciable improvement. For the past six years, Tucker's asthma spray has been used with moderate symptomatic relief. Infantile eczema which appeared during his first year cleared when he was two. At puberty, eczema recurred and has persisted in mild form since. Slight dermatitis was present over both malar regions, and on the neck when he was first seen. The nasal mucous membranes were pale, boggy, and appeared typically allergic. The sinuses transilluminated well. Inconstant asthmatic whistles were heard over the chest. Heart sounds were normal with blood pressure 116/74, and vital capacity 3.5 liters. Mantoux test with 0.1 mg. old tuberculin was positive. Sputum was negative for tubercle bacilli.

He was found by skin testing and clinical observation to be sensitive to house dust, feathers, dog, cat, and horse dander, grass and fall pollens, and to alternaria and hormodendron spores. He has since been given perennial treatment with dust, pollen, and mold antigen, with improvement in his asthma, although occasional attacks have continued. In April 1943, he was seen after mild asthma of two days' duration. He had worked all day and although he did not appear ill, his face was somewhat flushed and his temperature was found to be 103°. No abnormal physical signs were discovered on chest examination. The next day his temperature was normal. Similar febrile episodes occurred in May, October, and December of that year, usually preceded by a day or two of increased asthma. On January 2, 1945, he reported that seven days previously he felt flushed and chilly when he reached home from work. That evening, his temperature was 103° and at 4:00 A. M. he had a drenching sweat, followed by normal temperature thereafter. After one day at home, he returned to work feeling quite normal. No unusual chest findings were present on January 2, and on January 4, apart from some diffuse emphysema, the lungs were roentgenologically negative.

June 25, 1945, he reported that there had been considerable coughing

throughout the preceding day and some irritation remained in his throat. No abnormal chest findings were present. There was slight redness of the posterior pharyngeal walls, and his temperature was 99.4°. He was given 15,000 units of penicillin aerosol nebulized by oxygen at 3 liters per minute. One hour later, he reported a chill and temperature of 103.6°. When admitted to the hospital four hours later, his temperature was 103.2° and the leucocyte count was 14,800 with 88 per cent neutrophiles and 2 per cent eosinophiles. Chest roentgenogram at this time showed a walnut size infiltration in the anterior portion of the right lung at the level of the fifth rib. No abnormal physical signs were detected over this area or elsewhere, and in spite of the fever he did not appear ill. The next day his temperature was 99.0° and the leucocyte count was 17,550 with 72 per cent neutrophiles and 1 per cent eosinophiles. Because this attack resembled previous ones in all respects and because he did not feel ill, he returned home. The following morning his temperature was 99° and was normal thereafter. Roentgenogram on July 2 showed the area of involvement to be almost clear. No blood count was made on this date, but on July 25 the leucocyte count was 7,300 with 5.5 per cent eosinophiles.

Case II: T.G., a boy who was seen first in 1937 at the age of four, had perennial rhinitis and recurrent attacks of asthma. Infantile eczema began at six months and persisted until he was about a year and a half old. Characteristic asthmatic breath sounds were present and the nasal mucosa was boggy and typically allergic in appearance. Skin tests showed sensitivity to house dust, alternaria, and the pollens of grass and ragweek. Dust precautions were instituted and perennial hyposensitization has been carried out with the antigens named with considerable general improvement. A bronchoscopic examination in 1940 showed only findings compatible with asthma. Occasional episodes of asthma usually have been associated with upper respiratory infections. Sputum examinations were repeatedly negative. Chest roentgenograms were normal apart from emphysematous appearing lung fields.

November 17, 1945, his mother reported much coughing and a temperature of 99.4° because of which sulfonamides had been given. Mild fever continued and on November 23, he entered the hospital. Chest roentgenogram on admission showed considerable infiltration about both hilar areas, extending on the left side for a considerable distance toward the periphery of the lungs. The temperature was 102°. There was normal resonance everywhere over the chest, with scattered asthmatic whistles present. Medium-sized inconstant sticky rales were heard in the left front chest at the level of the fifth interspace. Leucocyte count in the morning was 28,000 with 2 per cent eosinophiles. Sputum cultures on admission showed no unusual organisms. He was given 10,000 units of penicillin intramuscularly every three hours and in addition, he was given penicillin aerosol. His temperature continued to rise to 101 or 102° each afternoon. Roentgenogram on November 28 showed bilateral hilar infiltration which was now marked on the right side, and had somewhat subsided on the left. Intramuscular penicillin was discontinued November 30, when it was apparent that its use had resulted in no appreciable change. Throughout this time, his general condition had been good with no evidence of toxemia consistent with the pulmonary involvement present. On December 3, chest roentgenogram showed that the intrapulmonary infiltration had largely disappeared. The transitory and migra-

tory character of the roentgen shadows now made the diagnosis of Loeffler's syndrome quite certain. Mantoux test on December 3 was negative with 0.1 mg. tuberculin. Stool examination was negative for ova and parasites, and agglutination test for B. abortus was negative.

In early February, he had a frank upper respiratory infection and raised yellow, purulent sputum. Again March 30, he was seen with a reddened throat, a temperature of 101.6° and had scattered sibilant asthmatic rales in the chest. Fever continued for about a week and subsided after oral penicillin.

TABLE I

Blood counts in Case II during two episodes of pulmonary infiltration.

Date	*W.B.C.*	*Neut. Seg.*	*Neut. Stab.*	*Eos.*	*Mono.*	*Lymph.*
		PER CENT				
24 Nov. '45	28,500	75.0	8	2	2	13
26 " "	17,700	63.0	2	9	7	19
27 " "	18,250	73.0	5	4	5	13
28 " "	16,200	71.0	3	8	3	15
29 " "	17,150	50.0	6	12	6	25
1 Dec. "	12,400	58.0	6	9	7	20
2 Jan. '46	15,600	53.5	0.5	19	3	24
19 " "	11,200	51.0	2.0	17	6	24
21 Aug. "	7,200	50.5	2.0	11	0	35.5
23 " "	12,300	45.0	1.5	21.5	2	30
30 " "	12,850	54.0	2.0	17	2	25
7 Sept. "	8,800	45.0	1.5	18	3	32.5
21 " "	11,800	48.0	1.0	16	2	33

August 4, he had severe asthma which lasted three days during which he stayed in his room equipped with a pollen filter. On August 17, chest roentgenogram showed a new infiltration in the first and second interspace on the left side which remained without appreciable change in roentgenograms of August 23 and September 3.

Leucocytes on August 21 numbered 7,200 with 11 per cent eosinophiles, and 12,300 with 21.5 per cent eosinophiles on August 23. A sputum specimen on August 21 contained masses of purulent yellow material, with many eosinophiles and pus cells present, but no Curschmann's spirals or Charcot-Leyden crystals. No acid-fast bacilli were found and there was no growth of fungi on Saburaud's medium. Beta hemolytic streptococci, S. viridans, and N. catarrhalis were present in blood agar sputum cultures. Albumen (grade 2) was found in the urine August 23 with 1-2 R.B.C. and 4-6 W.B.C. per high power field. No casts were present. (Previous urine specimens in June and July were negative). Urine culture on August 23 showed a fairly heavy growth of hemolytic staphy-

lococcus aureous and colonies of alpha hemolytic streptococci and S. viridans.

Throughout this period he did not feel especially ill, although his general appearance was not good. The presence of hemolytic cocci in the sputum and urine during the course of this last episode are felt to indicate that bacterial allergy was responsible in part, at least, for the pulmonary infiltration observed. Unequivocal proof of causal relationship is, however, lacking.

Case III: J.A.B., developed typical ragweed hay fever in 1928 at the age of seventeen. Symptoms recurred each fall thereafter with increasing severity. During the winter of 1935, while installing a heating system in a burlap bag factory, asthma occurred for the first time. Wheezing stopped at once after he left the building and there was no recurrence of asthma until 1940 at the peak of the ragweed season. Nasal symptoms which at first were confined to the fall season, gradually became perennial, although maximum rhinitis continued to occur in the spring and fall. In mid-May 1942, he observed dyspnea and some wheezing while climbing stairs and a diagnosis of virus pneumonia was made by his physician. Chest roentgenogram a little later showed infiltration of the left apex. A Mantoux tuberculin test at this time was positive. Repeated sputum examinations including cultures were negative for tubercle bacilli. He remained at home for seven weeks with bed rest during the first three. Roentgenogram June 3, 1942 showed the lung fields clear.

Throughout the summer of 1942, he had considerable rhinitis and during the ragweed season there were ten or twelve sharp attacks of asthma. In November 1942, a preemployment roentgenogram showed infiltration of the right middle lobe. Sputum and gastric specimens were negative for tubercle bacilli. November 30, 1942, he entered Muirdale

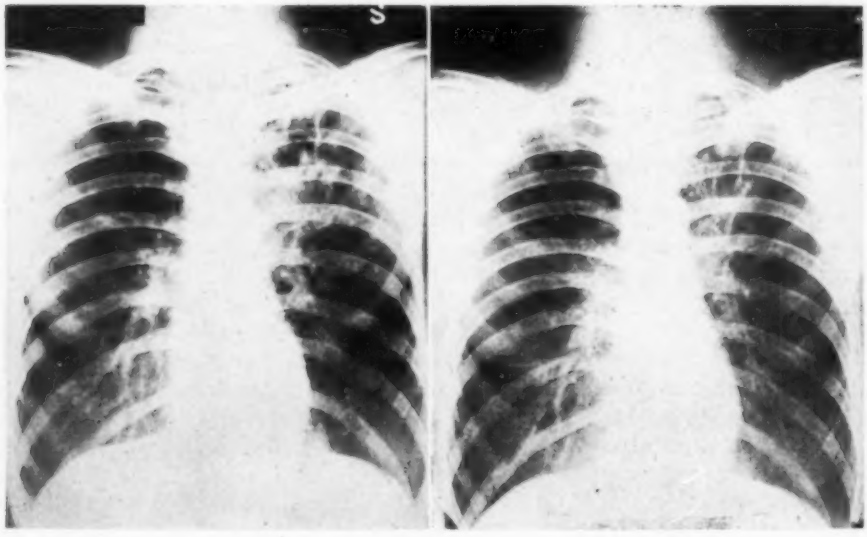


FIGURE 1 FIGURE 2

Figure 1, Case 3: May 25, 1942. Extensive infiltration, left upper lobe.—*Figure 2, Case 3:* June 3, 1942. Essentially clear. Questionable haziness in left subapical region.

Sanatorium where he remained until January 23, 1943. During this period, repeated sputum examinations showed many eosinophiles but were negative for tubercle bacilli; stool examinations were negative for ovae, amoebae, and parasites; muscle biopsy and skin test were negative for trichiniasis. A bronchoscopic examination showed nothing unusual and direct smear made from the secretions showed no fungi. During this period, there was a mild leucocytosis at times and eosinophiles fluctuated between 5 and 14 per cent. At no time did he appear ill and a tentative diagnosis of Loeffler's syndrome was made on discharge.

He later gave the additional story of occasional hives in childhood, and reported two injections of antitetanic serum in the past without reaction. His mother had been subject to headaches, a maternal uncle had allergic rhinitis, and his five-year-old son has hay fever. Cutaneous reactions were present to tree, grass, and fall pollens, to alternaria spores, and to house dust. With the exception of tree pollens for which no passive transfer tests were made, circulating reagins were demonstrated for these antigens and for wheat and rye. He has had perennial hyposensitization with the indicated inhalant allergens for the past three years with definite clinical improvement. However, there have been occasional mild asthmatic attacks and he has had several episodes of pulmonary infiltration sometimes preceded by malaise, cough, and increased asthma, but at other times infiltrations have been discovered on a routine roentgen recheck of the chest in the absence of any accompanying symptoms.

During the height of the 1943 ragweed season, there was slight asthma which continued into October. On November 26, after spreading marsh hay over his garden, he had a sharp attack of asthma which lasted for several days. On December 9, he had generalized grippe-like aching and some pain under the left shoulder. Medium-sized moist rales were present at the end of inspiration in the left infraclavicular region. His tem-

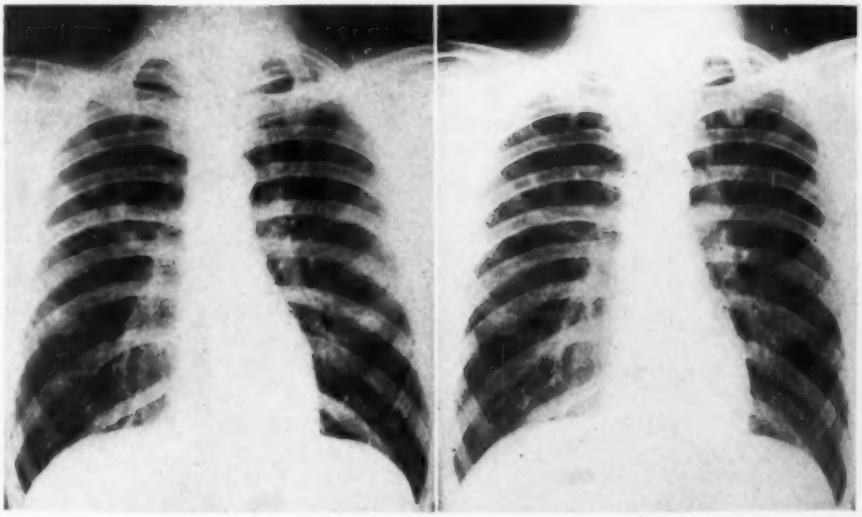


FIGURE 3 FIGURE 4

Figure 3, Case 3: Jan. 4, 1943. Infiltration in right mid-lung field. Sharply circumscribed, grouped nodules.—*Figure 4, Case 3:* May 22, 1943. Lung field clear.

perature was 100.4°, and the leucocyte count was 8,550 with neutrophils 77.5 per cent and eosinophiles 3.5 per cent. Roentgenogram at Muirdale Sanatorium December 11 showed dense infiltration in the left hilar region at the level of the second and third ribs anteriorly. Roentgenogram on December 18 showed marked clearing, and another December 24 showed still further improvement. During this episode, a single sputum specimen was reported positive for tubercle bacilli after culture and quinea pig inoculation. Since that time, repeated sputum cultures and guinea pig inoculations have all been negative for tubercle bacilli.

Roentgenogram April 18, 1944, showed a new and rather extensive infiltration in the right upper lung field with some residual fibrosis still visible in the left infraclavicular region. Leucocyte count was 6,950 with 22 per cent eosinophiles. Sputum culture was again negative for tubercle bacilli. Except for very slight wheezing which occurred on the night of April 25, there were no clinical symptoms during this period of pulmonary infiltration. No rales or physical signs were disclosed on examination other than those demonstrated in the roentgenogram, and he continued to work throughout this period without subjective feeling of illness. In the fall of 1944, considerable asthma, September 16 to 18, coincided with high atmospheric concentrations of alternaria spores, and subsequently, occasional mild wheezing continued throughout October. On November 20, he felt congestion in the chest and coughing increased. November 22, his temperature was 100.4°, but no abnormal chest findings were detected. Roentgenogram November 24 showed a wedge-shaped density in the left lower lung field with the apex directed laterally from the left heart border. Leucocyte count was 8,800 with 68 per cent neutrophiles and 10 per cent eosinophiles.

In the spring of 1945, occasional mild wheezing occurred during the

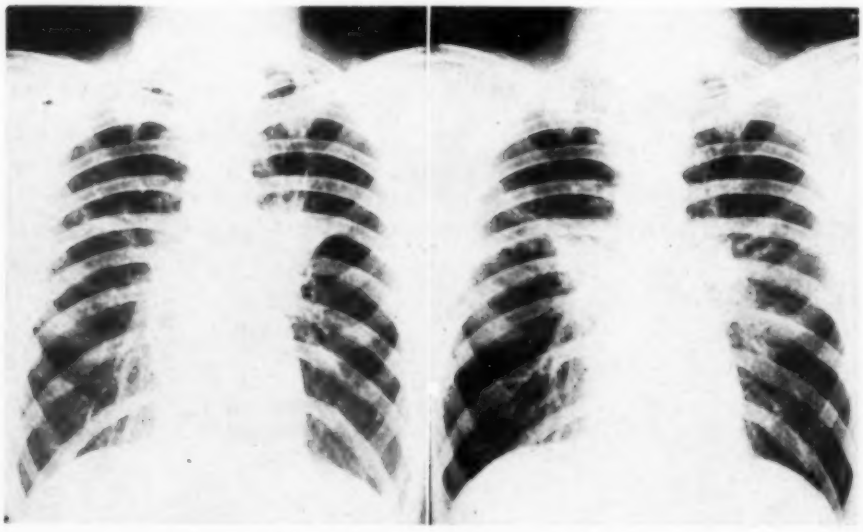


FIGURE 5 FIGURE 6

Figure 5, Case 3: Dec. 11, 1943. Dense infiltration in the left hilar region at level of second and third ribs anteriorly.—*Figure 6, Case 3:* Mottled infiltration in the right apex. Increased hilar density on each side. Slight increased density in left mid-lung field.

grass season. On May 16, excessive dust exposure while unpacking dusty materials caused coughing and increased wheezing which required ephedrine for relief. No subsequent symptoms were observed but on June 8, roentgenogram showed a new infiltration in the mid portion of the right lung, with considerable fibrosis still present in the upper left hilar region. Repeated sputum cultures on July 9 and August 3 were negative for tubercle bacilli and guinea pigs inoculated at this time were likewise negative when killed on September 14. Leucocyte counts on March 7, July 7, and August 22, 1945 were 12,500, 7,800, and 8,750 respectively with corresponding eosinophile count of 10, 13, and 12.5 per cent. Sedimentation rates at one hour on these dates were 15 mm., 16 mm., and 2 mm., respectively.

On September 15, 1945, chest examination was essentially negative. Roentgenogram at this time showed a rather extensive infiltration involving the upper lobe on the right side. Fibrosis in the left upper still remained, and there had been no change in appearance of the left hilus. A sputum specimen at this time was again negative for acid-fast bacilli by concentration test, and by culture. The leucocyte count on September 19 was 7,400 with 11.5 per cent eosinophiles. Roentgenogram on January 16, 1946, showed essential clearing of the lesion in the left upper chest with only a minor amount of scarring in this area represented by a few fibrotic strands extending into the first interspace. In the right lung, a fan-like infiltration extending from the hilus into the lower part of the right lobe still remained, but was less marked than in September. The leukocyte count January 22 was 9,000 with 16.5 per cent eosinophiles. On July 9, his general condition was good, the chest examination was normal, the leucocyte count was 6,050 with 0.5 per cent eosinophiles, and the sedimentation rate was 13 mm. at one hour. Sputum specimen at this time was negative for acid-fast organisms both by concentration test and cultures.

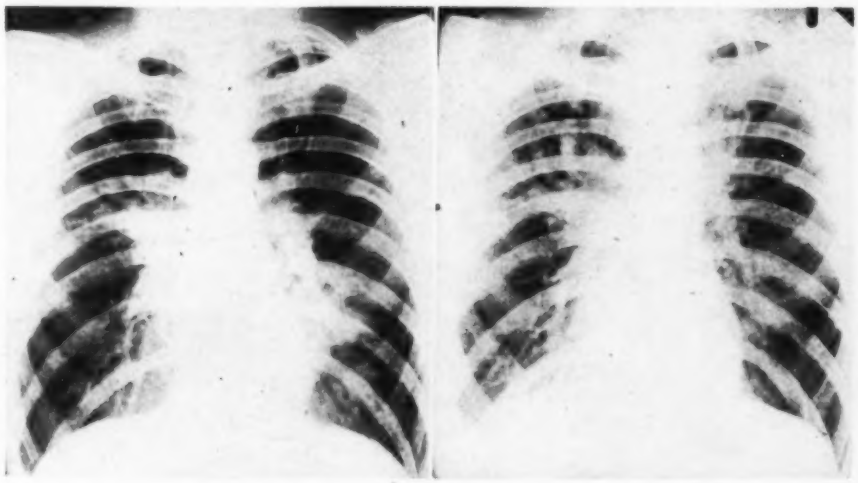


FIGURE 7 FIGURE 8

Figure 7, Case 3: Nov. 24, 1944. Apices clear. Hilar densities increased. Wedge shaped shadow in the left lower lung field.—*Figure 8, Case 3:* June 8, 1945. Very marked clearing of right middle lobe. Increased hilar markings remain, especially on the left.

COMMENT

Transitory pulmonary infiltrations conforming to Loeffler's criteria occurred in each of three patients reported. Multiple sensitivities to extrinsic inhalant allergens were demonstrated in each. In the first patient, repeated fulminating febrile episodes occurred without accompanying evidence of toxemia and with a recovery too prompt to be consistent with bacterial invasion. A transitory pulmonary infiltration was demonstrated by roentgenogram during one such episode. In both of the last two patients, bacterial sensitivities at times may have acted as the trigger mechanism for an allergic infiltration. The first recognized infiltration described in detail in Case II probably was initiated by such bacterial antigens, although no specific organisms could be incriminated. The later infiltration was accompanied by evidence of respiratory and urinary tract infection which may well have been responsible for the infiltrate. In the present state of our knowledge, control of such intrinsic or idiopathic factors is exceedingly difficult. In the third patient, it is apparent that extrinsic factors contribute materially to the clinical picture, and infiltrations often were related directly to massive exposure to such extrinsic allergens as dust, mold spores, or pollens. However, in view of the positive Mantoux test and of the single positive sputum reported, it is entirely possible that tuberculin may be an intrinsic antigen in this patient although clinical evidence of active tuberculosis continues to be lacking. The persistent and increasing fibrosis seen in the roentgenograms is adequate evidence that irreversible changes are developing in this patient which may well be of the type associated with hyperergic vascular damage such as described by Harkavy. Hyposensitization and avoidance of recognized extrinsic offenders has resulted in very definite general improvement and it is suggested that the irreversible process might well proceed with greater rapidity were it not for control of the recognized extrinsic sensitivities.

SUMMARY

1. Three cases of Loeffler's syndrome are discussed. In each instance, atopic rhinitis and asthma were present and sensitivities to multiple inhalant allergens were demonstrated.

2. Presumably, polyvalent bacterial or intrinsic sensitivities were present in all three patients in addition to recognized extrinsic sensitivities. It is difficult and often impossible in the presence of multiple sensitivities to incriminate any single antigen as the cause of an episode of allergic pulmonary infiltration.

3. In two of the patients described, pulmonary infiltrations were

characteristically transitory. In the third patient, although early infiltrations were of fleeting character, repeated attacks have left increasing residual fibrosis and evidence of irreversible damage. It is believed that irreversible changes after allergic pulmonary infiltrations are especially apt to occur when so-called intrinsic or bacterial sensitivities are present.

RESUMEN

1. Se discuten tres casos del síndrome de Loeffler. Todos ellos presentaban rinitis atópica y asma, y en cada uno se demostró sensibilidad a múltiples alergenos de inhalación.

2. Se supone que en todos los tres pacientes existían sensibilidades bacterianas polivalentes o intrínsecas, además de las sensibilidades extrínsecas reconocidas. Cuando se presenta sensibilidad múltiple es difícil, y a menudo imposible, incriminar a un solo antígeno como causante de un episodio de infiltración pulmonar alérgica.

3. En dos de los pacientes descritos las infiltraciones pulmonares fueron característicamente transitorias. En el tercer paciente, aunque los infiltrados precoces fueron de un carácter efímero, repetidos ataques dejaron una creciente fibrosis residual y signos de daño irreversible. Se cree que alteraciones irreversibles consecutivas a infiltrados pulmonares alérgicos tienden a ocurrir especialmente cuando existen las llamadas sensibilidades intrínsecas o bacterianas.

REFERENCES

1 Loeffler, W.: "Zur Differential Diagnose der Lungeninfiltrierungen. II Ueber flüchtige Succedan-Infiltrate (mit Eosinophilie)," *Beitrage zur Klin. der Tuberk.*, 79:368, 1932.

2 Loeffler, W.: "Die flüchtigen Lungeninfiltrate mit Eosinophilie," *Schweizerische Med. Wochenschr.*, 66:1069, (Nov. 7) 1936.

3 Zweifel, E.: "Cutaneous Tests with Ascarides Extract in Temporary Eosinophilic Pulmonary Infiltrates," *Helvetica Med. Acta*, 11:117, (April) 1944.

4 Slowey, J. F.: "A Case of Transient Successive Pulmonary Infiltration (Loeffler's Syndrome) Associated with Trichiniasis," *Ann. Int. Med.*, 21:130, (July) 1944.

5 Hoff, Amanda and Hicks, Mason: "Transient Pulmonary Infiltrations. A Case with Eosinophilia (Loeffler's Syndrome) Associated with Amoebiasis," *Am. Rev. Tuberc.*, 45:194, (February) 1942.

6 Randall, T.: "Eosinophilic Pneumonitis (Loeffler's Syndrome) with Response to Emetine," *Brit. J. Tuberc.*, 39:37, (January) 1945.

7 Elsom, K. A., and Ingelfinger, F. J.: "Eosinophilia and Pneumonitis in Chronic Brucellosis: A Report of Two Cases," *Ann. Int. Med.*, 16:995, (May) 1942.

8 Engel, D.: "Ueber eine eigenartige anaphylaktische Erkrankung der Lunge," *Beitrage zur Klinik der Tuberk.*, 87:239, (December 18) 1935.

9 Ellis, R. V., and McKinlay, C. A.: "Allergic Pneumonia," *J. Lab. and Clin. Med.*, 26:1427, (June) 1941.

10 Vaccarezza, R. F., and Parlovsky, A.: "Bronchopulmonary Allergy Produced by Gold Salts: Loeffler's Syndrome with Disseminated Foci and Micronodulary Picture," *An. Catedea de pato. y. clin. de la tuberc.*, 7: 164, (June) 1945.

11 von Meyenburg, H.: "Das eosinophilie Lungeninfiltrate. Pathologische Anatomie und Pathogenese," *Schweizerische med. Wochenschr.*, 72: 809, (July 25) 1942.

12 Harkavy, J.: "Vascular Allergy—Pathogenesis of Bronchial Asthma with Recurrent Pulmonary Infiltrations and Eosinophilic Polyserositis," *Arch. Int. Med.*, 67:709, 1941.

13 Klopstock, A., and Steinitz, H.: "An Epidemic of Eosinophilic Erythredema, Possibly Early Filariasis," Letters, Palestine, June 1, 1945, *J. Am. Med. Assn.*, 129:144, (Sept. 8) 1945.

14 Bayley, E. C., Lindberg, D. O. N., and Baggenstoss, A. H.: "Loeffler's Syndrome: Report of a Case, with Pathologic Examination of Lungs," *Arch. Path.*, 40:376, (November-December) 1945.

Endothelioma (Mesothelioma) of the Pleura*
(Presentation of a case treated surgically)

LEW A. HOCHBERG, M.D., F.A.C.S., F.C.C.P.
ISRAEL G. EPSTEIN, M.D., F.C.C.P., and
MORRIS PERNIKOFF, M.D.
Brooklyn, New York

Endothelioma of the pleura is a rare disease but even less common is the instance in which it is amenable to surgical extirpation. A case in which such a tumor was completely removed is to be presented in the following.

Controversy exists as to the origin of these tumors. Klemperer and Rabin (1931), Banyai and Grill (1933), Stout and Murray (1942), and Saccone and Coblenz (1943) believe they arise exclusively from the endothelial lining of the pleura; Herxheimer and Reinke (1914), and Grossek (1932) believe they arise from the endothelium lining the pleural lymphatics; Frankel (1892), Aschoff (1936), and Ewing (1940) place the origin of these tumors in the pleural endothelium; Robertson (1924), and Willis (1938) state that these tumors are secondary to primary neoplasms elsewhere as within the lung and finally, Fischer (1931), and Scheidegger (1936) subscribe to the view that these tumors arise on the basis of aberrant rests of pulmonary epithelium within the pleura. Whatever their origin, it is generally agreed that they arise in the pleura and that the term "Mesothelioma" would be a more inclusive term. Saccone and Coblenz suggest the compromise term "Pleuroma" until such time when more is known about these tumors.

Until comparatively recent times, it was taught that all pleural endotheliomata have a multicentric origin in the pleura and that they grow diffusely. Recent studies by Klemperer and his coworkers and by Stout and Murray (1943) indicate that there are apparently two forms of such tumors. One grows diffusely along the pleura causing pleural thickening and invades the underlying tissue—lung, diaphragm, thoracic wall, etc., and either does not metastasize or involves the mediastinal lymph nodes. The second form is a circumscribed tumor which grows as a relatively benign process until late in the disease and then extends beyond its confines to the surrounding tissues. Stout and Murray state: "one of these is distinctly of an epithelial appearance and consists of

*From the Department of Thoracic Surgery, Bushwick Hospital, Brooklyn, New York.

solid cords or hollow tubes of cells which sometimes seems to secrete a mucinous material. The other tissue assumes the appearance of a fibrosarcoma; spindle cells arranged in bundles, often with reticulin or collagen fibers between them. The epithelioid elements sometimes have suggested the hyperplastic proliferation of vascular endothelium when the cells assume a rounded or polygonal form, but more often seem truly epithelial with cuboidal or cylindrical cells arranged about lumens. The localized tumors, on the other hand, do not have the epithelioid elements but seem more like fibroma or fibrosarcoma."

Forty-three cases of endotheliomata of the pleura were collected from the literature in a series of 42,614 autopsies—a necropsy incidence of 0.10 per cent. At the Brussels St. Jean Hospital, Ley (1929) encountered nine cases in 2,000 autopsies—an incidence of 0.45 per cent, whereas Seydel (1910), at the Pathologic Institute of Munich, found but three cases in 10,829 necropsies, an incidence of 0.03 per cent. The disease occurs twice as frequently in males (146 cases) as in females (78 cases) and is about equally as frequent on the right side (59 cases) as on the left side (54 cases).

The subject of this report was a 54 year old Italian woman whose chief complaints were pain in the chest, asthenia and blood tinged sputum. About five years before the patients admission to the Bushwick Hospital, she began to notice intermittant dull aches in the left infrascapular region. During the first two or three years the pains were not remarkable. For the next two years the pains became more constant but not

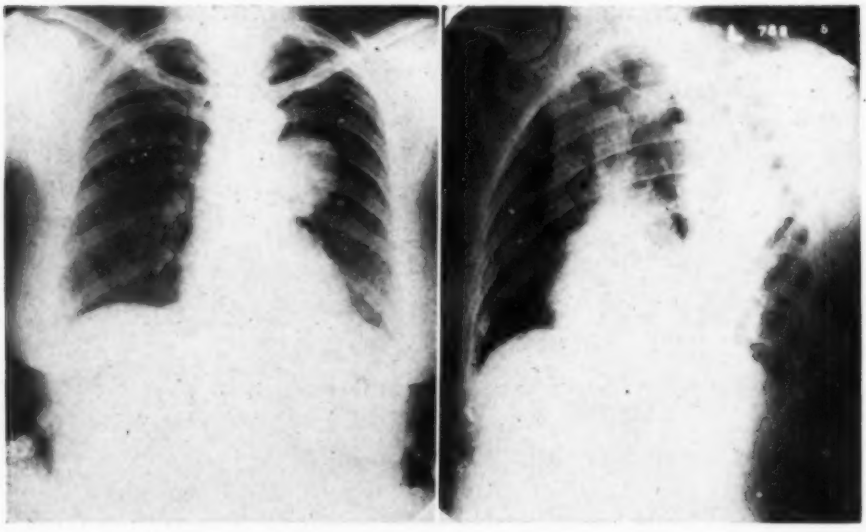


FIGURE 1 FIGURE 2

Figure 1: Roentgenogram of the chest showing a well circumscribed mass in the left parahilar region.—*Figure 2:* Roentgenogram of the chest in the left oblique position revealing the posterior position of the neoplasm.

necessarily more severe than previously. About three years before her admission to the hospital, she noted that the pain was becoming more severe, especially during the winter months. About the same time, she began to have cough with mucopurulent expectoration. At times, the sputum was blood tinged—this was most common when the patient developed a "cold".

Physical examination revealed a well developed, apprehensive woman. Except for the abnormal physical signs in the thorax, the physical examination revealed nothing unusual. Except for a rapid rate, the heart too presented nothing abnormal. The right lung was negative to percussion and auscultation. The left hemithorax was dull to percussion over the middle posterior third. Anteriorly, there was dullness to heavy percussion only, over the corresponding area. In the area of impaired percussion (posteriorly) the breath sounds were distant and bronchial in character—at times, there was a fleeting friction rub in the left scapular region.

Laboratory studies revealed an essentially normal blood count and a normal blood sugar and protein level. The Wasserman reaction was negative. The electrocardiogram was interpreted as being within normal limtis. Roentgenographic examination of the chest revealed a solitary spherical mass about two inches in diameter, in the posterior mediastinal part of the left lung field suggestive of a benign new growth (Figures 1 and 2). Bronchoscopic examination about ten days before the patient entered this hospital failed to reveal any disease in either lung. Preoperative diagnosis was benign tumor of the left lung.

At the time of surgical intervention on May 9, 1946, there was found a large, well demarcated neoplasm between the two lobes on the left side. As the two lobes were separated, a tumor arising from the left lower lobe was encountered. The neoplasm was well circumscribed and appeared embedded in the upper posterior part of the dark, discolored lower lobe. As the visceral pleura was split proximal to the tumor, it was noted that the part of the pleura over the tumor was thickened

FIGURE 3 FIGURE 4

Figure 3: Tumor after its removal, measuring 5x4x4 cms.—*Figure 4:* Tumor split open to show its consistency.

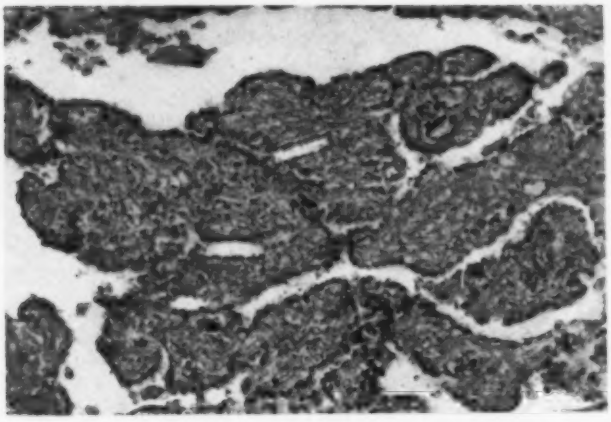


FIGURE 5: Photomicrograph of one region of the neoplasm. Within the papillary structure of the stem there are several large, flat cells. (180x).

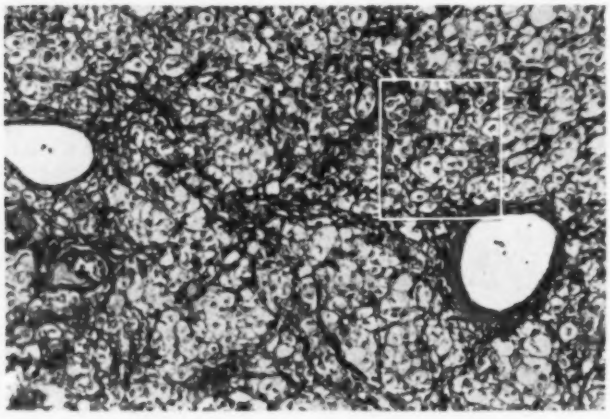


FIGURE 6: Photomicrograph of another region of the neoplasm. The cells resemble the endothelium of a blood vessel. (180x). The area delineated by the square is shown in Figure 7.

FIGURE 7: Photomicrograph of one part of the field shown in Figure 6. (850x).

and fused with the neoplasm. A proximal plain of clevage was then developed and the tumor freed of the visceral pleura. It was then noted that the neoplasm was embedded in the lung but did not appear to take origin from the substance of the lung. At one point (about half inch in diameter), the tumor was fused to the lung tissue necessitating the removal of the adherent part of the lung (Figures 3 and 4).

The pathologist's report was as follows: The specimen consists of a well encapsulated tumor measuring 5x4x4 cm. On its outermost surface, there are strands of fibrous tissue. Just deep to this, there are many ecchymotic areas and thin vessels. On section, one notes a solid tumor which is composed of yellowish and hemorrhagic areas with a few dilated reddish areas at the periphery. Microscopic study of the tumor tissue (Figures 5, 6 and 7), showed a rather diffuse growth of papillary projections the outer layer of cells being of a low cuboidal character with large hyperchromatic nuclei. Within the fibrous stalk, there are a number of large irregular cells with multiple hyperchromatic and irregular nuclei. In other sections, there is a diffuse growth of polygonal shaped cells having a rather pale granular cytoplasm with vesicular nuclei. There are also present a number of vascular channels throughout the tumor varying in size from a small capillary-like structure to large blood channels lined by endothelium. In some sections, there are areas of hyalinization. The histologic picture suggests the probability of blood vessel origin.

Diagnosis: Angioendothelioma of the pleura.

SUMMARY

A brief resumé of the subject of endothelioma of the pleura is presented with the report of a case in which surgical removal was carried out.

RESUMEN

Se presenta un breve resumen sobre el tema del endotelioma de la pleura y se informa sobre un caso en el que se llevó a cabo la extirpación quirúrgica.

REFERENCES

1 Aschoff, L.: *Path. Anatomie,* Vol. 2, Jena: Gustav Fischer, 1936.

2 Banyai, A. L., and Grill, J.: "So-Called Pleuropulmonary Endothelioma," *Am. Rev. Tuberc.,* 27:193, 1933.

3 Birnbaum, G. L.: "Primary Mesothelioma of the Pleura," *Quart. Bull., Sea View Hosp.,* 4:462, 1939.

4 Ewing, J.: "Neoplastic Diseases," *W. B. Saunders Co.,* Phila., 1940.

5 Fischer, W.: "in Henke-Lubarsch," *Handb. d. Spez. path. Anat. u. Hist.,* Vol. 3, pt. 3, Berlin: J. Springer, 1931.

6 Frankel, A.: "Ueber primaeren Endothelkrebs (Lymphangitis proliferativa) der Pleura," *Berl. Klin. Wchnschr.,* 24:497, 1892.

7 Grossek, R.: "Die Deckzellen der serosen Haute und ihre primaren malignen Geschwülste," *Ztschr. f. Krebsforsch.,* 35:435, 1932.

8 Herxheimer, G., and Reinke, F.: "in Lubarsch," *Ostertag Ergebn. d. allg. Path. u. Path. Anat.,* 16:225, 1914.

9 Klemperer, P., and Rabin, C. B.: "Primary Neoplasms of the Pleura," *Arch. Path.,* 11:385, 1931.

10 Ley, R. A.: "Apropos des tumeurs dites 'endotheliomes' de la plevre," *Bruxelles-med.,* 10:57, 1929.

11 Robertson, H. E.: "Endothelioma of the Pleura," *J. Cancer Research*, 8:317, 1924.

12 Saccone, A., and Coblenz, A.: "Endothelioma of the Pleura," *Am. J. Clin. Path.*, 13:186, 1943.

13 Scheidegger, S.: "Ueber die sogen. Endotheliome der Pleura," *Ztschr. f. Krebsforsch.*, 42:93, 1935.

14 Seydel: "Ueber Operabilitat von Lungen-und Pleuratumoren," *München. med. Wchnshr.*, 77:452, 1910.

15 Stout, A. P., and Murray, M. R.: "Localized Pleural Mesothelioma," *Arch. Path.*, 34:951, 1942.

16 Willis, R. A.: "Metastatic Deposit of Bronchial Carcinoma in Hydrocele Misdiagnosed 'Endothelioma' of Serous Membranes," *J. Path. and Bact.*, 47:35, 1938.

An Unusual Aortic Aneurism
Followed for Ten Years

W. G. HARTNETT, M.D., F.C.C.P., and
C. S. BEATTY, M.D.*
Muskogee, Oklahoma

Little new can be added to the knowledge of aneurism. Galen knew external aneurisms well. Lancisi, in 1728, wrote a monograph on their etiology. Morgagni described the symptoms and morbid anatomy as early as 1761.

There has come under our attention a most unusual case, which has been followed for a period of ten years. Norris and Fetterolf[1] reported a similar case in 1917. Meerloo,[2] too, described a case in 1929, in which the aneurism eroded both the sternum and the vertebral column. Any text book will describe erosions of the sternum complicating aneurism, but in this case, not only did the aneurism erode the sternum, but perforated it, and projected as a horny tumor on the anterior chest wall.

L. L. was brought to the hospital February 7, 1936, at which time he complained of smothering spells, edema of his extremities, productive cough, shortness of breath, worse at night, and ascites. His pulse was 90, blood pressure: 130/110. Examination showed his heart enlarged in all

*From the Veterans Administration Hospital, Muskogee, Oklahoma.

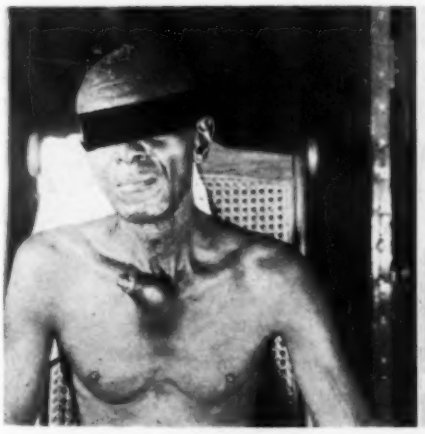


FIGURE 1

FIGURE 2

Figure 1: The appearance of the aneurism on September 20, 1945, at which time cornification of the skin had begun.—*Figure 2:* The size of the horny growth can be appreciated as it is seen being held up by the patient. Likewise, in this illustration, the aneurismal wall can be seen with the blood oozing from its surface. This photograph was taken the day of the patient's death.

diameters, and on auscultation there was a systolic murmur, which was faint in character, located at the base, transmitted down the left sternal border. The heart tones were weak, distant and muffled. The Wassermann test was 100 per cent positive; the EKG showed a left ventricular preponderance; the x-ray film showed the transverse diameter of the heart to be 18.5 cm.; diameter of the aorta: 9.8 cm. Fluoroscopic examination of the chest showed a pulsating, saccular tumor at the arch of the aorta, and the diagnosis of aneurism was made. There was a history of penile sore in 1909. He was treated with bed rest, digitalis and salyrgan, and after compensation was established he was given antiluetic treatment, consisting of potassium iodide and mercury, and a course of bismuth. After 102 days in the hospital he was discharged considerably improved.

Four and a half years later he returned to the hospital, on September 4, 1940. He was complaining of soreness in chest, aching in back, smothering spells and weakness. The soreness in his chest, which he had been having for fifteen years, he stated was gradually getting worse. His cardiac diameter was now measured at 19.5 cm. On this admission his blood pressure was 150/85, and the aorta now measured 10.5 cm. A diastolic murmur had appeared at the base. His Wassermann test had become negative, and he was given a diagnosis of aortic insufficiency.

After twenty-seven days he was again discharged as improved, and was admitted for the third time on February 4, 1945. At this time he complained of shortness of breath and swelling of the lower extremities. The x-ray film showed the transverse cardiac measurements to be 22.5 cm.; his EKG showed many ectopic beats, widening of the QRS, suggesting left bundle branch block. Examination showed an expansile

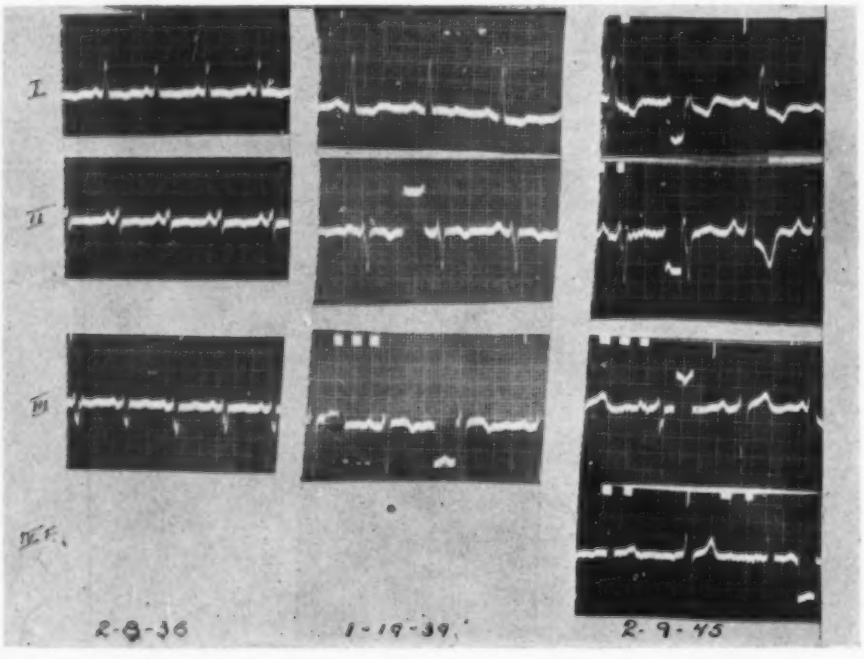


FIGURE 3: Electrocardiograms taken on February 8, 1936, on January 19, 1939, and February 9, 1945.

FIGURE 4 FIGURE 5 FIGURE 6

Figure 4: Shows the appearance of the chest on his first admission when the diagnosis of aneurism and cardiac failure was first made.—*Figure 5:* Shows six weeks later, when his compensation was established and his passive congestion had fairly cleared up.—*Figure 6:* Left oblique: shows erosion through the sternum.

mass, measuring 8 cm. in length and 6 cm. across its base, protruding upward and outward, to the right of the upper part of the sternum.

After four months he was again discharged as improved. However, he was admitted again on August 9, 1945, his final admission. He had again developed swelling of his legs and shortness of breath. He became compensated after three days of treatment, and was ambulant and comfortable until March 20, 1946, when the aneurism ruptured and the patient expired.

Three days prior to the patient's death, the horn projecting from his sternum dropped off, and the aneurism could be seen pulsating and oozing blood from its wall.

SUMMARY

An unusual appearing aneurism is presented, together with a ten year study. It is offered only as unusual and an interesting case.

We are indebted to Mr. Charles Edwards of the X-Ray Department for the photography.

RESUMEN

Se presenta un aneurisma de raro aspecto, junto con un estudio de diez años. Se ofrece este caso solamente como raro e interesante.

Le agradecemos la fotografía al Sr. Charles Edwards del Departamento Radiológico.

REFERENCES

1 Norris, G. W., and Fetterolf, G.: "Frozen Sections From a Case of Sternum-Eroding Aortic Aneurism," *Tr. Assn. Am. Phys.*, 32:420-434, 1917.

2 Meerloo, A. M.: "Aneurism of the Aorta with Erosion of the Sternum and Vertebral Column in a Patient with Dementia Paralytica," *Nederlandsch Tijdschrift voor geneeskunde*, 1:536, (Feb. 2) 1929.

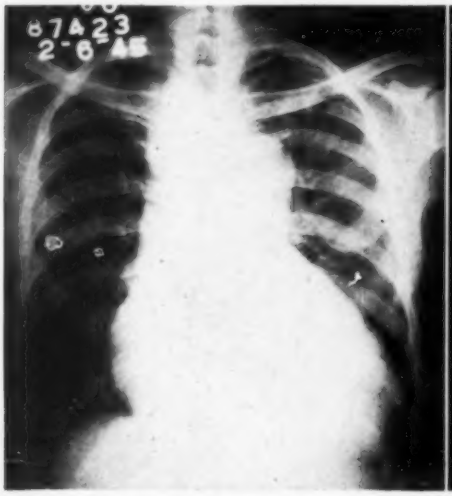


FIGURE 7

FIGURE 8

Figure 7: Shows the appearance on his last admission. The aneurism may be seen coming through the sternum and projecting up under the skin.—*Figure 8:* Shows left oblique: showing left heart enlargement and the perforation of the sternum at the time of his last admission.

Thoracoscopy and Pneumonolysis*

J. D. MURPHY, M.D., F.A.C.S., F.C.C.P.**
Oteen, North Carolina

In 1934, Moore[1] reviewed the literature on closed intrapleural pneumonolysis and published the results in a collected series of 2,114 cases. In 1944, Goorwitch[2] in an exhaustive study analyzed the results and complications of the operation in forty-eight of his own patients, as well as in operations on 5,114 patients published by various authors in the literature from 1933 to 1943. These two series, representing two successive decades, are remarkably similar in essential results and complications. There are some operators in each group whose results are so good that their analytical judgment is open to question. For instance, the incidence of postoperative pleural effusion ranged from 3 to 25.5 per cent. One operator reported 13 per cent of postoperative empyemas but only 1.3 per cent of bronchopleural fistulae. Others have an unusually high percentage of complications but have doubtless salvaged a number of unsuccessful pneumothoraces by attacking adhesions their more conservative colleagues have avoided.

The present trend toward early abandonment of an unsuccessful pneumothorax has made thoracoscopy almost a mandatory procedure in a substantial percentage of cases. Nearly all operators agree that only by thoracoscopy can an accurate determination as to the possibilities of severing adhesions be made. By thoracoscopy also much other information of value in determining the adequacy of the pneumothorax can be obtained. The presence or absence of subpleural tubercles, of blebs and bullae, and the degree of inflammatory pleural reaction are all matters that may be weighed before a final decision as to maintenance of the pneumothorax can be made.

It is my feeling that thoracoscopy should be used more frequently than at present, since it is a relatively simple procedure with few complications, and yields high dividends in essential information.

Pneumonolysis, on the other hand, is a highly technical and potentially dangerous procedure which has too often resulted in disaster to the patient through the operators zeal to improve an

---

*Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the author.

**Veterans Hospital, Oteen, North Carolina.

unsuccessful pneumothorax. Since we now have in phrenic nerve operations combined with pneumoperitoneum, and in thoracoplasty, two safe and successful methods of treatment, it behooves the operator to "stop-look-and consider" the possibility of empyema and bronchopleural fistula before attempting to free a border line type of adhesion. In any event, the operation should not be performed, unless facilities and personnel are available for an open thoracotomy, if hemorrhage should make it necessary.

Pneumoperitoneum has gained such an ascendancy over pneumothorax at the Oteen Veterans Administration Hospital in the past eighteen months that we are being called upon less frequently to perform a pneumonolysis.

Our attitude has been conservative as to pneumonolysis, but enthusiastic as to thoracoscopy. After intrapleural visualization, we have doubtless considered inoperable many adhesions, which bolder souls would attack without hesitation. The favorable results obtained here with pneumoperitoneum and our success with thoracoplasty, however, have tipped the scales toward conservatism in many questionable adhesions.

Form July 1943 to July 1946, we have done 234 thoracoscopies on 224 patients. On 88 of these patients, 96 pneumonolyses were performed. This represents an operability rate of 41 per cent.

The thoracoscopies have been singularly free of complications. In a few cases, a transient elevation of temperature or accumulation of serous pleural fluid occurred. One case developed a temporary bronchopleural fistula, the consequent pneumothorax had to be decompressed and the lung reexpanded. His disease subsequently attained an arrest. One case developed a tuberculous empyema, and died three months postoperatively. It is probable that in any 224 pneumothoraces without thoracoscopy, an equal number of complications would have been encountered.

In 48 operations, or 50 per cent of the total number of pneumonolyses, a complete division of all the adhesions visualized was performed. In 23 operations, or 24 per cent, an incomplete division was done. By this it is meant that one or more adhesions were completely divided but that some adhesions remained which it was not considered possible to divide. In 25 operations, or 26 per cent, there was a partial pneumonolysis, one or more adhesions being partially severed but for reasons of safety, or bleeding, it was felt dangerous to attempt to complete the division.

Following 38 operations, or 39 per cent of the group of 96 pneumonolyses, the sputum remained positive. In 17 of these patients, however, there was sufficient contralateral disease to account for the positive sputum. Twenty-nine per cent were negative before the operation and remained negative. In 36 operations, that is in

40 per cent of the 88 patients, the sputum was converted from positive to negative. Thus, in 69 per cent of these operations a negative sputum was either present initially or resulted from the lysis.

The double puncture technique with the electrocautery was used routinely. The complications encountered were as follows:

1) Hemorrhage, 5 per cent. In two cases the hemorrhage was of only moderate extent and resulted in no morbidity. In three cases there was bleeding of 500 cc. or more. In one young colored male with a rather broad adhesion, a large vessel, presumably an intercostal, was injured so that there was a flow of blood which arched in a crescent half way across the pleural cavity. The base of the parietal attachment of the adhesion was cauterized and the bleeding diminished but did not cease. An immediate thoracotomy was performed and the operation completed as an open pneumonolysis. This patient did well following the operation and subsequently his sputum was converted. The second case with a hemorrhage of over 500 cc. did not bleed at the operation. A thoracentesis was done the next day for fluid and blood obtained. A thoracoscopy was done and the point of division of the adhesion visualized. No active bleeding was present. He recovered uneventfully following two more thoracenteses. The third case was one of the patients who died and he will be discussed under a subsequent heading.

2) Purulent effusion occurred in two operations, or 2 per cent of the cases. One of these was a mixed empyema and one of pure tuberculous type. Both of these patients died some time after the operation. They will be discussed in detail later.

3) Fever of over 100 degrees followed 29 operations, or 30 per cent of the procedures. This fever persisted over an average of six days postoperatively. In 21 operations, or 22 per cent, fluid either increased or a fresh accumulation appeared subsequent to the pneumonolyses. These were serous accumulations of a transient nature and resulted in no morbidity.

4) Bronchopleural fistulae were present in 2 per cent, or followed the operations.

5) Deaths: Four patients died in the 308 days following operation. Two of these were not attributable to pneumonolysis. One died with a mediastinal abscess subsequent to a bronchoscopic accident, and one died of a massive hemorrhage from a contralateral hilar cavity six months subsequent to the pneumonolysis. The death of two patients, 2 per cent of the operations, were attributable to pneumonolysis. One white male, age 48, had a pneumonolysis with a long band adhesion divided at the chest wall on August 4, 1943. His temperature was elevated the first

postoperative day to 102.6 degrees and remained over 100 degrees for sixty days. The temperature then ranged from 98 degrees to 99.6 degrees until December 1, 1943, when it became elevated to 102 degrees and showed a daily elevation thereafter until his death on January 7, 1944, five months after the operation. At the time of the pneumonolysis, 800 cc. of turbid, amber fluid was withdrawn through a catheter inserted in the thorax. August 17, 1943, 550 cc. of thin, green fluid was removed. From 200 to 500 cc. of thin, light green fluid was removed at weekly intervals up until the time of his death. On August 4, 1943, the fluid was negative for acid-fast bacilli and pyogenic organisms. On August 16, 1943, acid-fast bacilli were present. September 14, 1943, acid-fast bacilli and staphylococci were demonstrated. While this death occurred five months postoperatively and while pus was not actually demonstrated, the presence of tubercle bacilli and staphylococci in the fluid indicated that a mixed infection was present and the death must be attributed to the operation. Actually the presence of turbid, amber fluid at the time of the operation would cast some doubt as to whether or not this chain of events might not have occurred even without the operation. The second death attributable to the operation was that of a 27 year old white male who had partial pneumonolysis on July 25, 1944. His temperature was elevated to over 100 degrees from the first postoperative day until the time of death. The first day postoperatively, a positive pressure was found in the pneumothorax space. On three occasions from July 25 to September 2, the date of his death, sanguineous fluid was removed from the pleural cavity. A contralateral cavity appeared. This death must also be attributed to the operation due to the fact that the positive intrapleural pressure the first day postoperatively indicated the presence of a bronchopleural fistula. Sanguineous fluid removed on three occasions also indicated that hemorrhage of more than moderate degree had occurred.

SUMMARY AND CONCLUSIONS

1. In a series of 234 thoracoscopies from 1943 to 1946, ninety-six pneumonolyses were performed.

2. Thoracoscopy has proved to be a safe, simple and informative procedure, singularly devoid of serious complications.

3. Sixty-nine per cent of the pneumonolysis operations resulted in a negative sputum or made possible the continuation of a pneumothorax in a previously negative case.

4. The serious complications following the 96 pneumonolyses were: Hemorrhage, 5 per cent; Empyema, 2 per cent; Death, 2 per cent.

5. These results are in general agreement with the averages of the series of Moore and Goorwitch, but at variance with some of the individual authors of those series.

RESUMEN Y CONCLUSIONES

1. En una serie de 234 toracoscopias llevadas a cabo de 1943 a 1946, se ejecutaron noventa y seis neumonolisis.

2. Se ha comprobado que la toracoscopia es un procedimiento inofensivo, sencillo e informativo, particularmente exento de complicaciones graves.

3. El sesenta y nueve por ciento de las operaciones de neumonolisis dieron como resultado un esputo negativo o permitieron la continuación del neumotórax en casos previamente negativos.

4. Las complicaciones graves que siguieron a las 96 neumonolisis fueron: Hemorragia, 5 por ciento; Empiema, 2 por ciento; Muerte, 2 por ciento.

5. Estos resultados están generalmente de acuerdo con los promedios de las series de Moore y Goorwitch, pero están discordes con algunos de los autores individuales de esas series.

REFERENCES

1 Moore, J. A.: "Intrapleural Pneumonolysis," *J. Thoracic Surg.*, 3:276, 1934.

2 Goorwitch, J.: "Closed Intrapleural Pneumonolysis," *J. Thoracic Surg.*, 13:223, 1944.

Unilobar Tuberculosis Treated by Lobectomy

ROBERT D. DUNCAN, M.D., F.A.C.S.*
Surgeon, U.S.P.H.S., and

WAYNE W. CARPENTER, M.D.
Surgeon, U.S.P.H.S.

Springfield, Missouri

The surgical treatment of various diseases of the chest is making rapid strides. The techniques involved are contributing to a greatly decreased mortality rate from chest diseases. The lessening of the operative risk stimulated the authors to study the further extension of surgical treatment in pulmonary tuberculosis. This study was initiated somewhat over a year ago and is reported in this article. The premise advanced is that lobectomy possibly may have a place in the treatment of some unilateral tuberculous lesions, as a means of eradicating the tuberculous focus for individual cure of the disease. The various comments of authorities prominent in the field of tuberculosis concerning lobectomy are familiar to the authors. Most of the arguments cited are against such a procedure. However, the authors felt, and hoped, that the operative risk of lobectomy had decreased to the point where a guarded trial of such a procedure was justified. We are happy to say that our hopes were justified and we feel that in no case has our surgical treatment harmed the patient. In fact, although our series of lobectomies is small and not statistically of value, we feel that definite patient improvement, rehabilitation, and perhaps cure or arrest of the tuberculosis has resulted in every instance. It is admitted that this must be a preliminary report, as the time elapsed following operation—about one and a half years—is not sufficient without further follow-up study for final evaluation. The results so far, however, are quite striking. This consecutive series of five cases is reported for its face value.

Classification of Lesions

In a recent article[1] the minimal lesion was divided into several groups on a basis of study of 469 such lesions. These groups are as follows:

Exudative Group: Those showing pneumonic infiltration, but without progression to cavitation.

Exudative-Productive Group: In this group there were exudative

---

*Diplomate, American Board of Surgery. From the Tuberculosis and Surgical Services of the U. S. Medical Center, Springfield, Missouri.

and productive processes associated with organization and healing, but the lesions were considered unstable.

In the above two groups progression of the lesion occurred in 33 per cent of white patients and in 49 per cent of non-white patients within one year, and in 49 per cent of the white and 61 per cent of the non-white patients within five years.

Productive-Fibrotic Group: Those showing foci consisting of either tuberculous granulations or fibrosis, or both.

Fibro-Calcific Group: Those showing calcareous or cheesy calcareous foci with advanced anatomical healing.

In the last two groups above, the five year incidence of progression of the diseases was 5 per cent for white patients and 14 per cent for non-white patients. In the cases showing progression, most of the patients were stated to have advanced or far advanced disease.

The effect of conservative therapy in treatment of minimal tuberculous lesions is difficult to evaluate. There are no definite data available for evaluation, because of the marked variants that occur—selection of cases, selection of treatment, lack of uniformity of institutional care, differences within the series of cases themselves, such as percentages of the different types of minimal lesions, racial differences and other factors. In the previously mentioned 469 cases the progression of disease was found in 33 per cent of white patients and in 49 per cent of non-white patients. Other series reported are lower.[2] These authors report a rate of progression for 33 per cent of all patients.

In the selection of cases for lobectomy, the distinction between lesions likely to progress and those not likely to progress can be made if the character and progressive tendencies of the minimal lesions to be considered are kept in mind.

The unilobar lesion is differentiated from the minimal lesion by anatomical location. A unilobar lesion must be limited to one side. It may be a minimal lesion. It may show progression by the development of cavitation or local spread.

Selection of Cases for Lobectomy: Most cases of minimal tuberculosis selected for lobectomy today show bronchial ulceration and stenosis, atelectasis, bronchiectasis, other complications, or are those that have failed to respond to previous surgical methods. This places a heavy responsibility on lobectomy. That such a procedure has been able to retain favor under these circumstances is, in itself, a certain commendation. A fair trial as compared to conservative therapy would consist of its application in cases without complications. The series of cases herein reported falls into the latter group. We have not operated upon minimal lesions *per se,* but we selected for lobectomy five unilobar lesions with evidence

of cavitation. As a group the cases showed definite evidence of progression rather than healing. We eliminated cases with bronchoscopic evidence of stenosis or ulceration. We felt that the complete eradication of the focus of infection was not possible in such cases.

Criteria for the Selection of Cases: (1) The tuberculous lesion must be unilobar (limited to one lobe and not evidenced elsewhere). This determination is difficult and cannot be accomplished in all cases. However, if all diagnostic studies indicated that only one lobe were involved, it is felt that the percentage of error would be very small. In a case not included in this series, an exploratory thoracotomy was done because of what appeared to be a tuberculous involvement of the left upper lobe with markedly enlarged hilar lymph node, possibly lymphoma or carcinoma. At operation the lesion of the upper lobe was found to be tuberculous and a caseous nodule about 1½ to 2 cm. in diameter was found in the medial portion of the apex of the lower lobe. The upper lobe was removed. The nodule broke down following complete expansion of the lower lobe and formed a cavity which now has a thick wall. The patient's sputum is negative and no spread had occurred in the lower lobe, so apparently the removal of the upper lobe did no harm. This case is not included in this reported series, because at the time of operation the lesion was recognized as not being unilobar. Eighteen months postoperatively, this patient feels well and has gained in weight. The sputum is negative.

(2) The second criterion for lobectomy is evidence of lesion progression under conservative therapy, such as cavitation, local spread or hemorrhage.

(3) There must be no bronchial ulceration or tuberculous granulations. In the series reported an attempt was made to eradicate all evidence of tuberculosis. Bronchial ulceration or extension usually means that the surgical incision (of the bronchus) extends through or leaves some definite tuberculous tissue behind.

(4) The candidate for lobectomy should not be over forty years of age. This requirement reduces the operative risk. It may be that this is not necessary as long as the patient is otherwise in good condition. There is, of course, an operative risk with any procedure, but if the risk can be kept low, perhaps by limiting the procedure to a lower age limit, it may be considered justified.

Principles Involved in the Procedure: (1) It is realized that pulmonary tuberculosis, while localized in the lungs, is a systemic disease in that it has general systemic reactions. These reactions may be revealed by change in the blood count, sedimentation time, spread of disease to other organs, etc. Such reactions are true of other diseases and the removal of all of the evident foci of infec-

tion of disease is advocated and carried out in many conditions. This same principle can be applied to certain types of tuberculosis. In a good many diseases, the elimination of the major focus of infection of disease allows the body to overcome the residual disease. With lobectomy, the time necessary for cure or arrest of tuberculosis may be greatly reduced. Most of our lobectomy patients are permitted to be up from seven to twelve days postoperatively, but they are maintained for three months on a regime of regulated rest, exercise and diet, similar to that carried out for cases treated conservatively. The first case in our series was an overly enthusiastic patient who, in spite of instructions, frequently would be found playing baseball and indulging in other athletics. He started this about the third of fourth week postoperatively. His course has been excellent, however, and at the present time (two years postoperatively), he shows no evidence of tuberculosis. There is no deformity or impaired physical power and the patient feels well. There is no pain or dyspnea and there is no deformity or muscular weakness, as sometimes occurs with thoracoplasty.

(3) We would like to put forward the premise that lobectomy does not entail any greater risk of complications or spread of disease than do conservative methods of therapy. With pneumothorax the complications that occur are infection, empyema, air embolism and bronchial obstruction. Reactivation of the lesion following reexpansion of the lung occurs in a number of instances. Frequently, adhesions prevent adequate collapse and pneumonolysis with its added hazard, is necessary. The danger of complications following lobectomy is no greater and, actually, appears to be less. The risk of spread to the other lung due to compensatory overactivity of the contralateral lung would not appear to be greater with lobectomy than with pneumothorax or other procedures. The slight overexpansion of the remaining lung on the operated side in lobectomy may increase the possibility of activation of any remaining or overlooked lesion. A phrenic interruption at the time of operation may decrease this hazard. Careful examination of the chest to rule out involvement of any other portion of the lung will guard against such activation. In the one case mentioned previously, in which the lower lobe was involved, there was no spread or extension of the disease postoperatively. Pneumothorax and thoracoplasty do not remove the tuberculous lesion. Instead, it is relaxed so that extension of the process is possible and does occur postoperatively in a percentage of cases.

(4) Another important factor in selection of patients for lobectomy is the degree of operative risk and mortality involved. In a group of cases chosen according to the criteria listed previously,

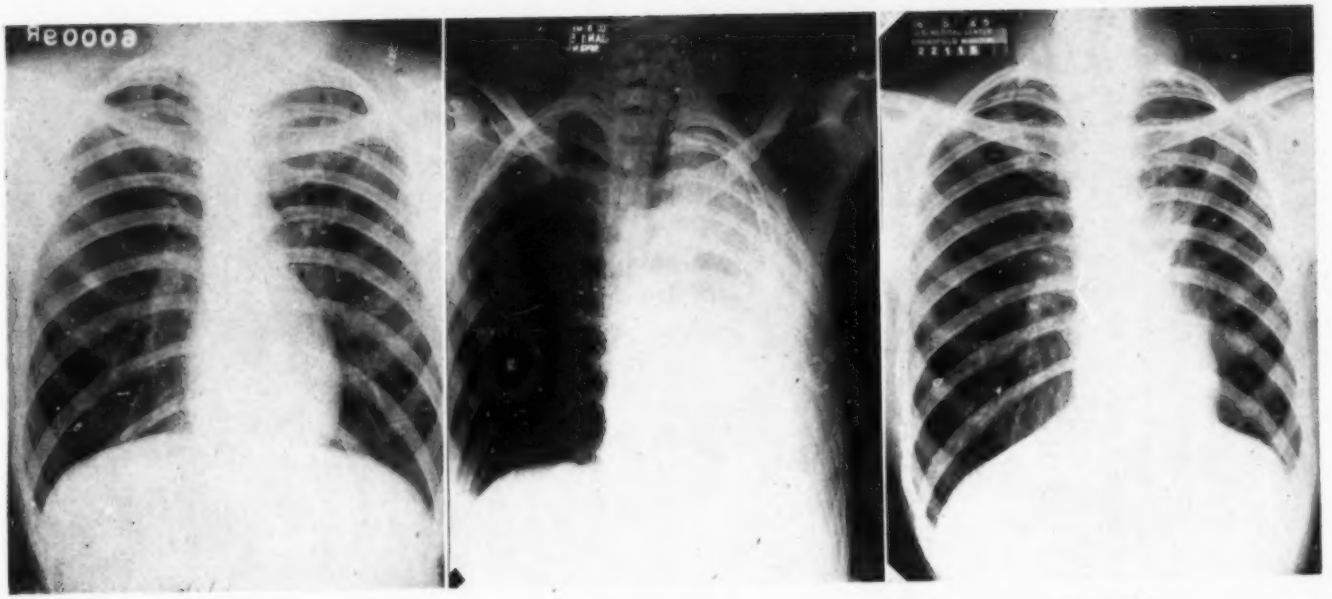


FIGURE 1 FIGURE 2 FIGURE 3

Figure 1, Case 1: Preoperative chest film.—*Figure 2, Case 1:* Immediate postoperative film.—*Figure 3, Case 1:* Follow-up film.

the mortality rate should be very low, and this will be the case if the patients are in experienced hands. Even for cases complicated with bronchiectasis, the mortality rate reported in a large series of cases is as low as three per cent or less. In the series reported here there were no fatalities, although the series is small. This is approaching the mortality rate in thoracoplasty.

CASE REPORTS

Case I: C. S., a 25 year old white male, was admitted to the Medical Center on October 7, 1944. During a tuberculosis survey in September 1944, a minimal tuberculous lesion was found involving the left upper lobe. Subsequently, an infiltrative lesion developed at the level of the first interspace, with an area of rarefaction suggestive of a small cavity. Artificial pneumothorax was begun at the referring institution. Physical examination on admission to this hospital revealed a well developed and fairly well nourished adult white male in no apparent distress. The chest was clear to auscultation and percussion. No rales were elicted. Urinalysis was essentially negative on admission. RBC, 6,430,000; Hgb., 16.4 gms.; WBC, 8,550, with a normal differential count. A standard film of the chest on admission revealed a beginning collapse of the left lung with a small amount of fluid at the left base. The right lung and the left lower lobe were normal in appearance. Sputum examinations were consistently negative. A gastric washing on December 7, 1944 was negative for tubercle bacilli. Blood sedimentation rate was 7 mm. per hour (Cutler) on October 12, 1944 and 6 mm. on January 8, 1945. The patient received routine tuberculosis care and pneumothorax refills. A good collapse of the left lung was demonstrated on a film taken December 14, 1944. He began expectorating blood-streaked sputum on November 16, 1944. Hemoptysis continued from that date until November 23rd. A moderately severe hemorrhage occurred on November 17th. Hemoptysis recurred on December 11, 1944 and continued in varying amounts until January 5, 1945.

On January 10, 1945 a left upper lobe lobectomy was performed under intratracheal ether and oxygen anesthesia. At the time of operation the

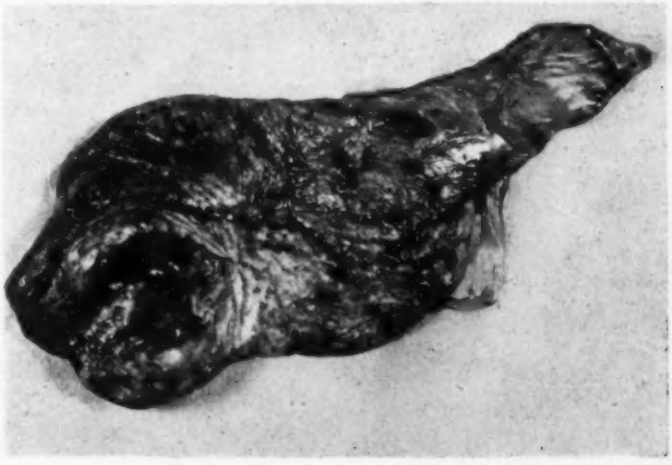


FIGURE 4. Case 1: Gross specimen.

apex of the upper lobe was found to be attached to the parietal pleura by a broad adhesive band which contained lung parenchyma. Several miliary spots were present on the visceral pleura at the apex. Several firm, indurated areas were palpated within the lung. The pathological examination revealed tuberculosis of the left upper lobe with several areas of caseation and multiple foci of the disease throughout the lobe. The postoperative course was entirely uneventful. The patient was returned to the tuberculosis service on January 25, 1945 for further observation. The operative wounds were completely healed. He had no further complaints referable to his chest. Sputum examinations have remained negative. On August 6, 1945 the patient was transferred to another institution. Re-check x-ray films July 1946, show no evidence of tuberculosis. Blood sedimentation rate was 5 mm. per hour (Culter) on July 8, 1946. Gastric washing on the same date was negative for acid-fast bacilli.

Case II: H. W., 28 year old white adult made, was admitted to the Medical Center on April 16, 1944 with a diagnosis of pulmonary tuberculosis. He gave a history of having had pneumonia in 1941. Shortly thereafter a diagnosis of tuberculosis was confirmed. He received sanatorium care for a period of nine months in 1942. He was hospitalized in Atlanta in October, 1943 until his transfer to this hospital. While at Atlanta, he had a severe pulmonary hemorrhage in November 1943. Physical examination on admission revealed a well developed and well nourished adult white male. His weight was 186 pounds and his height 67 inches. The chest was essentially negative to auscultation and percussion. No rales were elicited. A standard film of the chest revealed the heart to be pulled somewhat into the left hemithorax. The diaphragm was smooth and the costophrenic angles clear. There was a tuberculous process corresponding to the left first, second and third interspaces anteriorly. There were drainage lines extending from these opacities to the hilus. The right

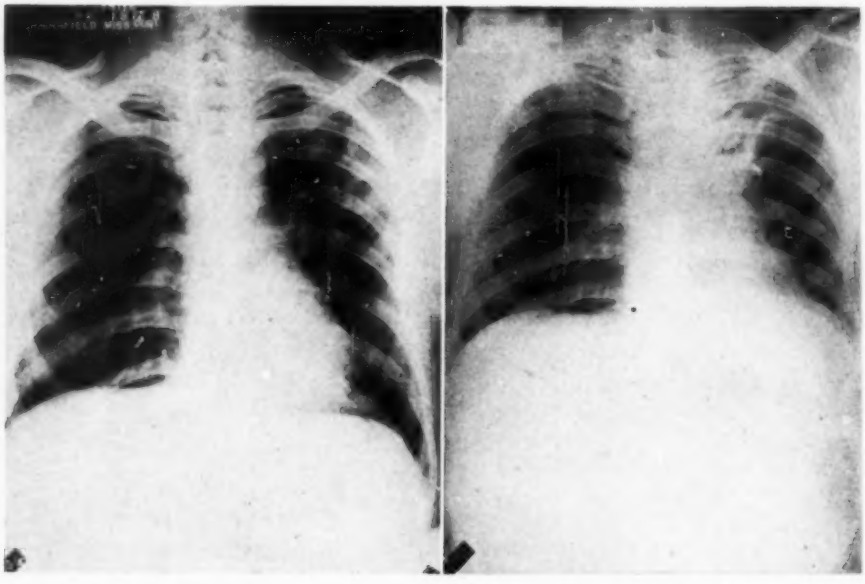


FIGURE 5 FIGURE 6

Figure 5, Case 2: Preoperative chest film.—*Figure 6, Case 2:* Follow-up film.

lung was normal in appearance. Urinalysis on admission was essentially negative. Examination of the blood showed: RBC, 5,680,000; WBC, 7,750; Hgb., 16.4 gm.; normal differential count. Blood sedimentation rate was 16 mm. per hour (Cutler). Kolmer and Kahn tests were negative. Sputum examinations were reported as negative for tubercle bacilli on April 22, 1944 and on May 15, 1944. A re-check x-ray film of the chest on July 10, 1944 showed renewed activity in the lesions previously described and, in addition, there was the appearance of a cavity at the level of the second interspace on the left. Sputum examinations were positive for tubercle bacilli on July 6, 1944. Artificial pneumothorax was begun on July 22, 1944. A film on July 29th demonstrated beginning collapse of the left upper lobe with the cavity slightly smaller and there was an adhesion demonstrated at the level of the second interspace. A film on October 14, 1944 revealed a poor pneumothorax with a small amount of air present. The cavity remained patent. There was an adhesion shown opposite the hilus and there were no doubt many adhesions preventing the collapse of the lung. The sputum remained positive in October 1944. An x-ray film on January 5, 1945 showed a better collapse of the lung; however, two cavities were now discernible which were not collapsed in spite of a good pneumothorax. The sputum remained positive in January and February. A re-check film on April 5, 1945 revealed a good collapse of the left lung, but the cavities were still apparent. There was no pleural effusion present. The right lung showed no evidence of tuberculosis. The patient received a total of 38 pneumothorax refills from July 22, 1944 to May 7, 1945.

On May 15, 1945 a left upper lobe lobectomy was performed. The pathological examination revealed tuberculosis of the left upper lobe, with a cavity 1½ cm. in diameter and four circumscribed white areas of necrosis in the medial portion. No change was noted in the peribronchial lymph nodes. The patient made an uneventful postoperative recovery and was returned to the tuberculosis service on June 2, 1945 for further observation. An x-ray film on January 8, 1946 revealed thickened pleura in the left apex. The right lung had escaped the disease, as had the lower portion of the left lung. The left hemidiaphragm was high. There was no evidence of tuberculous disease. Blood sedimentation rate was 6 mm. per hour (Cutler) on January 22, 1946. Sputum examinations postoperatively have been consistently negative for tubercle bacilli. Gastric washing on January 27, 1946, however, was reported as being positive. The patient has no symptoms referable to his chest. He has remained afebrile. The patient's weight on February 10, 1946 was 191 pounds. Frequent sputum examinations in April 1946 have been consistently negative for tubercle bacilli.

Case III: O. S., a 33 year old negro adult male, was admitted to the Medical Center on April 16, 1944 as a transfer from Atlanta with a diagnosis of pulmonary tuberculosis. On admission, the patient stated that in June 1943 he developed a productive cough, his sputum was blood-streaked and he began losing weight, the latter amounting to 13 pounds in a period of approximately four months. A few months later, he began having night sweats and sharp stabbing pains in the left lower chest which were aggravated by inspiratory efforts. He stated that his sputum was found to be positive for acid-fast bacilli in March 1944 while at the referring institution. The patient's weight on admission was 144 pounds and his height was 64 inches. Examination of the chest revealed dry,

crackling posttussic rales in the left apex to the fifth rib posteriorly and in the left supraclavicular region. There were dry rales occurring in the middle of inspiration in both bases anteriorly. Urinalysis was essentially negative; RBC was 4,950,000 with 15.9 gm. of hemoglobin; the white cell count was 7,400; differential: neutrophiles, 60; small lymphocytes, 22; large lymphocytes, 8; stabforms, 8; eosinophiles, 2 per cent. X-ray examination of the chest on admission revealed the heart and trachea to be pulled into the left hemithorax. The left apex was fairly clear. Corresponding to the second, third and fourth interspaces on the left, there was a large, soft density which represented an active infiltration. There were two areas of lessened density in this shadow which were suggestive of small cavities. Behind the third rib on the right, there was a soft density which represented a probable spread of the tuberculous disease from the left lung. Sputum examinations were negative for tubercle bacilli on April 22 and May 15, 1944. Blood sedimentation rate was 17 mm. in 60 minutes (Cutler). A re-check film on July 1, 1944 showed no evidence of spread of the disease. There was no definite evidence of cavitation. Sputum was positive on July 28, 1944. A film on October 13, 1944 showed no evidence of activity in the right lung. Small cavities were discerned in the left lung. Artificial pneumothorax was begun on the left side on October 30, 1944. A film on January 5, 1945 showed numerous adhesions in the left apex which were preventing a collapse of the upper lobe. There was no evidence of pleural effusion. Bronchoscopy was performed on May 4, 1945. There was some thickening of the carina. No mucous membrane irritation or ulceration was seen. There was no exudate or drainage from the bronchi on either side. Smears taken from the bronchi were negative for acid-fast becilli.

A left upper lobe lobectomy was performed on July 3, 1945. The patient's left upper lobe was entirely adherent to the parietal pleura in the region

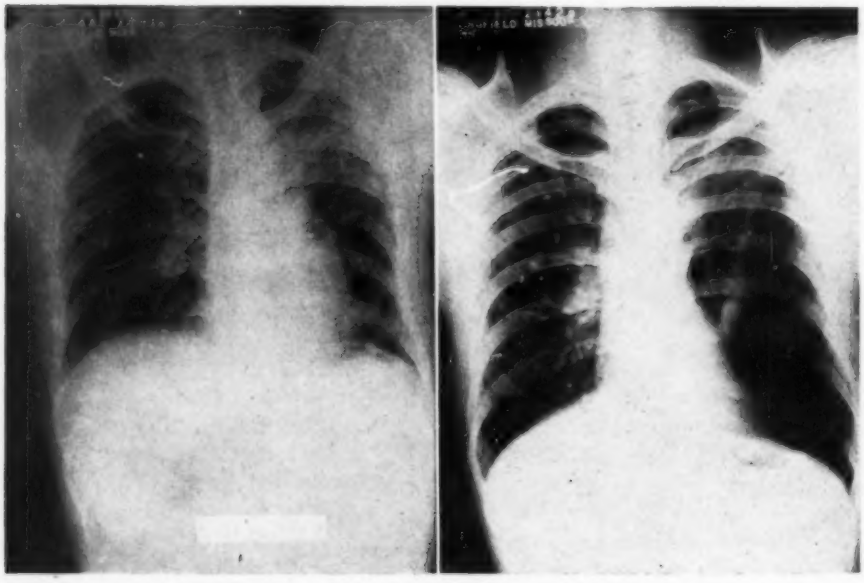


FIGURE 7 FIGURE 8

Figure 7, Case 3: Preoperative chest film.—*Figure 8, Case 3:* Results with pneumothorax.

of the mediastinum, apex, lateral wall and posteriorly, the only free portion being anteriorly. The left upper lobe was freed from its attachment to the parietal pleura with some difficulty and it was necessary to remove portions of the pleura of the chest wall along with part of the lung in certain areas. There was also a band-like adhesion from the lingula to the pericardium. Microscopic tissue examination revealed many nodular and caseous foci of tuberculosis throughout the left upper lobe and lingula. The postoperative course was essentially uneventful. X-ray examination of the chest on January 8, 1946 and June 3, 1946 showed the heart and mediastinum to be shifted into the left hemithorax. The greater portion of the left lung field was obscured by thickened pleura.

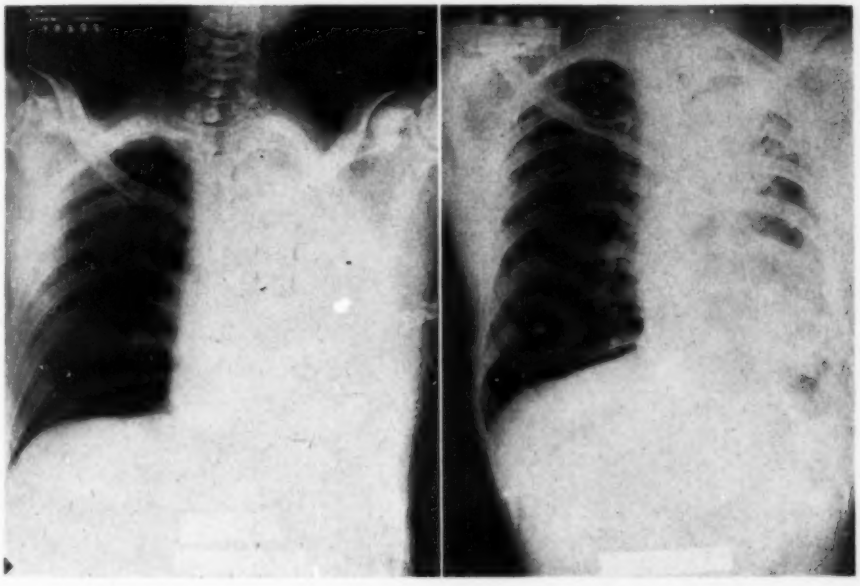


FIGURE 9 FIGURE 10

Fig. 9, Case 3: Immediate postoperative film.—*Fig. 10, Case 3:* Follow-up film.

FIGURE 11. Case 3: Gross specimen.

FIGURE 12

FIGURE 13

FIGURE 14

Figure 12, Case 4: Preoperative chest film.—*Figure 13, Case 4:* Immediate postoperative film.—*Figure 14, Case 4:* Follow-up film.

However, the left lung showed increased aeration as compared to a film taken on August 21, 1945. The right lung showed no evidence of tuberculous disease. The patient's admission weight was 144 pounds. Prior to operation the patient had a gradual weight loss. On July 1, 1945, two days preoperatively, his weight was 121 pounds. Thirteen days following lobectomy he weighed 114 pounds.

The sputum was negative on January 29, 1946. A guinea pig was inoculated with gastric washings on February 11, 1946. Necropsy of the animal on May 6, 1946 revealed no evidence of tuberculosis. Sputum examinations by the concentration method were negative on June 3, 1946 and July 17, 1946. Examination of a gastric washing on July 8, 1946 was negative for acid-fast bacilli. The patient weighed 122 pounds on July 14, 1946. He is afebrile and has no complaints referable to his chest.

Case IV: F. J. W., a 34 year old white adult male, was admitted to the Medical Center on May 5, 1945. In March of 1944, the patient volunteered to be infected with malaria at Atlanta. Prior to the project, a routine roentgenogram was made of his chest and tuberculosis involving the right lung was discovered. In February 1945 the patient began having dull pain in his right chest which was aggravated by respiratory efforts. He also had night sweats and a nonproductive cough. Weight loss was quite marked: from 156 to 138 pounds during the three-month period prior to admission to this hospital. Physical examination on admission revealed the patient to be a fairly well developed and nourished adult male, age 34, in no apparent distress. His height was 70¾ inches and his weight was 139 pounds. The chest was essentially negative on auscultation and percussion. No rales were elicited. The heart was normal and blood pressure was 126/74. There was a right rectus healed scar. X-ray examination on admission revealed the right apex to be filled with soft shadows down to the third interspace. There was the appearance of two small cavities at the level of the third interspace. A calcified primary lesion was present at the level of the second interspace on the left with calcified lymph nodes shown in the left hilar area. The disease on the right was thought to be active and probably progressing. Laminograph studies of the chest on May 14, 1945 revealed two moderate sized cavities beneath the third and fourth ribs posteriorly. No active disease was visualized in the lower right or in the left lung fields. Artificial pneumothorax was begun on May 21, 1945. On June 17, 1945, the patient began having moderate pulmonary hemorrhages, which continued for two days, and recurred on June 23, 1945. He continued to have moderate hemoptysis off and on until the time of operation. Sputum examinations were positive for tubercle bacilli on admission.

On July 5, 1945, a right upper lobe lobectomy was performed. At the time of operation, the right upper lobe was nodular in consistency and was adherent to the chest wall in its apical and medial portions. The right middle and lower lobes appeared to be free of disease, with the exception of the hilar portion of the middle lobe in which one moderate size calcified gland could be palpated. Microscopic tissue examination revealed a diffuse tuberculous involvement of the right upper lobe with several areas of cavitation. The postoperative course was essentially uneventful. The patient was returned to the tuberculosis service on July 18, 1945 for further observation. Periodic x-ray examinations of the chest postoperatively have shown the right lung to be completely expanded with no evidence of any parenchymal disease. The calcified area in the

FIGURE 15 FIGURE 16 FIGURE 17

Figure 15, Case 5: Preoperative chest film.—*Figure 16, Case 5:* Immediate postoperative film.—*Figure 17, Case 5:* Follow-up film.

left lung has shown no evidence of activity. The blood sedimentation rate three weeks postoperatively was 22 mm. in one hour (Cutler). This rate had dropped to 2 mm. on October 15, 1945 and was 3 mm. on January 22, 1946. Sputum examinations and gastric washings have remained negative following operation. A guinea pig was inoculated with gastric washings on March 28, 1946. Necropsy of the animal on May 6, 1946 revealed no evidence of tuberculous involvement. On July 17, 1946, sputum examinations were negative. The patient has remained afebrile postoperatively. The patient weighed 139 pounds on admission. His weight on February 10, 1946 was 150 pounds, a weight gain of eleven pounds during hospitalization.

Case V: D. L. A., a 31 year old white adult male, was admitted to the Medical Center from Atlanta on May 18, 1944. On admission, the patient gave a history of chest pain, gradual weight loss, productive cough and intermittent hemoptysis which began in February 1943. He was admitted to an Army General Hospital in California on May 27, 1943 for observation and treatment. X-ray examination of the chest at that time revealed moderately advanced pulmonary tuberculosis involving the upper lobe of both lungs. Sputum was positive for tubercle bacilli. The patient was subsequently admitted to Atlanta on October 30, 1943. X-ray examination at the time showed some infiltration in the right apex. There was no other evidence of active tuberculous disease. Sputum examinations were negative during the period of hospitalization at Atlanta. On admission to the Medical Center, chest roentgenograms revealed the hilar shadows to be somewhat increased and there were several calcified lymph nodes on the right. Sputum examinations were negative and the blood sedimentation rate was 4 mm. in one hour (Cutler). The patient remained under observation for a period of one year. On May 21, 1945 the patient complained of pleuritic pain in the right chest and had an elevation of temperature. X-ray examination revealed tuberculous infiltration with exudation and probable cavity formation in the upper portion of the right lung. The lower portion of the right lung and the left lung showed no evidence of activity. Sputum was positive for tubercle bacilli on May 30 and May 31, 1945. Laminograph studies on June 6, 1945

FIGURE 18, Case 5: Gross specimen.

revealed an oblong-shaped cavity in the right mid-clavicular plane which was seen best at the two and four centimeter levels (from the anterior chest wall) beneath the right second rib. Artificial pneumothorax was begun on June 15, 1945 and continued until the time of operation.

A right upper and middle lobe lobectomy was performed on July 10, 1945. At the time of operation, numerous adhesions were encountered. One broad and fibrous adhesion contained vessels extending from the chest wall. Definite cavitation in the upper lobe was determined by palpation. Several shotty, discrete nodular areas of small size were palpable in the right upper lobe. At the time of operation it was felt that there were some small miliary nodules present also in the middle lobe and for this reason the middle lobe was removed. However, subsequent examination has revealed that the middle lobe was free of disease. Microscopic tissue examination revealed tuberculosis of the right upper lobe with caseation and cavitation. There was no evidence of past or present tuberculosis of the right middle lobe. The first two days postoperatively the patient had a temperature elevation to 102 degrees and expectorated some thick mucous plugs, apparently due to atelectasis. He responded well to CO_2 and O_2 inhalations and penicillin. Temperature was normal the fourth day and remained so. A transitory ulnar neuritis was experienced for a few days following operation. This was undoubtedly due to the position of the right arm during the operation. Subsequent x-ray examinations of the chest showed the right lower lobe expanded and the diaphragm elevated, but no evidence of disease present. The left lung has remained clear. Bronchoscopic examination was done on August 27, 1945. Direct bronchial smears were negative for tubercle bacilli September 25, 1945. An x-ray report from the Veterans' Facility in February 1946 revealed no further evidence of disease. Sputum examination May 1946, done elsewhere, was reported to be positive. However, x-ray films in June 1946 show no evidence of further change or disease. The patient's weight was reported to be over 200 pounds. He was feeling quite well and was afebrile. On November 30, 1946 animal innoculation with sputum was reported negative.

COMMENTS

The cases presented and the surgical results obtained suggest that lobectomy may have a place in the treatment of the early lesion of tuberculosis in addition to pneumothorax and other procedures. Lobectomy should not be applied to every early or unilobar tuberculous lesion, since many of these lesions respond to conservative therapy. Patients with minimal lesions showing evidence of progression may be considered as possible candidates for lobectomy. The percentage of minimal lesions that will progress over a period of a year has been reported as 33 per cent or more. In the cases presented here, the progress of the disease appears to have been arrested in every instance. If this will hold true in a larger series of cases, lobectomy would appear to have advantages over the other procedures, such as pneumothorax, apicolysis, phrenic operations, open and closed intrapleural pneumonolysis, various treatments of cavities, and thoracoplasty. Admittedly

such procedures have a low mortality rate, but they do not always control the disease.

SUMMARY

1. Five consecutive cases of unilobar tuberculosis treated by lobectomy are presented with follow-up data.

2. The classification of patients with minimal lesions into different groups is mentioned. The value of careful selection of patients for lobectomy is considered and the criteria for this selection outlined.

3. The possible advantage of lobectomy in selected cases is discussed and compared with other methods of treatment.

4. It is felt that lobectomy has a place in the treatment of early tuberculosis, although a larger series of cases will be required to substantiate this fully.

RESUMEN

1. Se presentan cinco casos consecutivos de tuberculosis unilobular tratados por lobectomía, incluyendo los datos de la observación subsecuente.

2. Se menciona la clasificación de pacientes con lesiones mínimas en diferentes grupos. Se considera el valor de la selección cuidadosa de pacientes para la lobectomía y se bosquejan las pautas para esta selección.

3. Se discuten las posibles ventajas de la lobectomía en casos seleccionados y se la compara con otros tratamientos.

4. Se opina que la lobectomía tiene su lugar en el tratamiento de la tuberculosis precoz, aunque será necesaria una serie más extensa de casos para verificar completamente esta opinión.

REFERENCES

1 Reisner, D., and Downs, J.: "Minimal Tuberculous Lesions of the Lungs," *Am. Rev. Tuberc.*, 51:393-412, 1946.

2 Kruger, A. L., Potter, B. P., and Jaffin, A. E.: "Management of the Minimal Tuberculous Lesion," *Am. Rev. Tuberc.*, 46:50, 1942.

Treatment of Recurrent Spontaneous Pneumothorax with Gomenol*

L. H. HETHERINGTON, M.D., F.A.C.P., F.C.C.P.** and
G. E. SPENCER, M.D.***
Pittsburgh, Pennsylvania

Cajuput oil, or gomenol, deserves more recognition than it has received in the treatment of recurrent spontaneous pneumothorax. A survey of the literature of the past ten years finds this drug seldom mentioned, except to reject it as "uncertain" or "too caustic." However, we have evolved a safe and simple method to produce an obliterative pleuritis with gomenol, without the necessity of thoracoscopy or other operative procedure.

The mechanism of spontaneous pneumothorax has been thoroughly studied, and it is not the purpose of this paper to discuss etiology. To summarize, however, aside from disease in the lungs such as tuberculosis and silicosis, the cause is believed to be the rupture of an emphysematous bleb on the surface of the lung. Ornstein and Lercher[1] give an excellent review of the pathogenesis, and the reader is referred to this work for complete details. In the cases to be presented, one was of silicotic origin, the others idiopathic.

Many substances have been used in an effort to promote the formation of pleural adhesions. R. A. Young[2] used the patient's own blood, or 30 per cent glucose in combination with decompression. He also suggested the use of 5 per cent gomenolized paraffin as an oleothorax. Brunner[3] sprayed the lung fistula through a thoracoscope with substances to cause obliteration. Brock[4] used 5 to 10 minims of 10 per cent silver nitrate solution, and always thoracoscoped his patients prior to injection. Scrubbing of the parietal pleura with dry gauze has been tried, and tapes have been inserted into the pleural space to cause mechanical irritation.

Bethune[5] advocated the insufflation of ½ per cent iodized talc under thoracoscopic control, and rejected chemical irritants as useless, uncertain, or dangerous. Gowar[6] reviews a large number of substances in animal experiments, but agrees with Bethune on the use of poudrage.

---

*From the Tuberculosis League Hospital, 2851 Bedford Avenue, Pittsburgh, Pennsylvania.
**Assistant Professor of Medicine, University of Pittsburgh.
***Assistant Instructor of Medicine, University of Pittsburgh.

Gomenol has proved to be highly satisfactory when used by the method to be described. It is a volatile oil distilled from fresh leaves and twigs of *Melaleuca leucodendron*. It is a colorless or slightly yellowish liquid with a pleasant camphor odor.

The method of use is as follows. The patient is prepared by giving morphine sulfate 16 mg. (¼ grain) twenty minutes prior to treatment. The patient is placed on the unaffected side, and, using aseptic precautions, one cc. of gomenol in 5 cc. of 1 per cent novocaine is inserted into the pneumothorax space, the mid-axillary line in the fourth interspace being the site of choice. A 10 cc. syringe with an 18 gauge needle is used, care being taken that air can be drawn into the syringe before the gomenol is injected. There is usually a moderately severe pain reaction for several hours, which is controlled adequately by morphine sulfate 8 mg. (⅛ grain) every three to four hours for 24 hours. A moderate (100° - 102° F.) febrile reaction may occur on the first day.

A pleural effusion will begin to form in the first 24 hours, and the patient may complain of increased dyspnea. If too severe, air may be withdrawn until the intrapleural pressure is well on the negative side. In most instances the dyspnea will be eased without interference within 24 hours. Serial x-ray films will show rapid reexpansion of the lung, and the fluid tends to resorb rapidly. One injection is usually sufficient, but a second dose may be repeated in 3 days if the pleural reaction is not sufficiently great. Complete reexpansion will occur in two to four weeks.

Five cases are presented to show the effectiveness of this treatment. All had recurrent spontaneous collapses, one as a result of silicosis, the others of idiopathic nature. Ordinary methods of treatment failed to prevent recurrence of the condition.

All were engaged in non-strenuous occupations at the time of the pneumothorax. Four out of five were affected on the left side, which is characteristic, but contrary to published figures, three out of five occurred in women. The age range was from 21 to 50 years.

CASE REPORTS

Case 1: Mr. E. H., age 31 years. White male. Occupation: Meter reader. On January 15, 1937, while riding to work on a streetcar, the patient experienced a sharp knife-like pain in the left chest, producing a choking sensation and nausea. He was placed in bed by his physician and remained there for one week, then returning to work with occasional pain in the left chest on exertion. Increasing shortness of breath, chest pain, and exhaustion made him again consult his physician, when a diagnosis of spontaneous pneumothorax was made after x-ray examination.

On June 1, 1937, air was withdrawn from the chest, and 500 - 700 cc. were withdrawn twice weekly for several weeks. No re-expansion of the lung occurred, so the patient was admitted to the Tuberculosis League

Hospital on July 20, 1937. X-ray examination showed almost complete collapse of the left lung, with the exception of some apical adhesions. There was mediastinal herniation and dislocation of the heart to the right. Laboratory findings: Urine, neg.; RBC, 5,280,000; WBC, 8,450; Hgb., 84 per cent; Sputum, repeatedly negative for tubercle bacilli.

On July 21, 1937, a thoracoscopy was performed and there appeared to be a very slight perforation of the lung at the apex. Adhesions at the apex were partially dissected in an effort to make a better collapse and close the perforation. Closed drainage of the chest under a water seal was carried out following thoracoscopy, but the lung did not re-expand under this treatment. The symptoms were somewhat alleviated, however, and he was discharged on July 26 as improved, since herniation and dislocation of the mediastinal structure were not present at this time.

His symptoms recurred about a month later, and he was re-admitted to the hospital on September 8, 1937, for closed withdrawal of air. On admission, ¼ cc. of gomenol and 10 cc. of normal saline were injected in the left pleural space. One week later 1 cc. of gomenol and 10 cc. of normal saline were injected, and a week following this, the same dosage was repeated. There was continuous drainage of air under closed water seal for two weeks, following the injection of gomenol. He complained of acute pains in the side for one day after the first dose of gomenol. After two weeks, closed drainage was discontinued.

Serial x-ray films showed pleural effusion in the left pleural space and beginning re-expansion of the lung, so that by the time the patient was discharged on December 23, 1937, there was complete re-expansion of the left lung, a small amount of fluid at the left base, and some retraction of the mediastinal structures to the left. The lung fields remained clear. The patient gained eight pounds in weight while in the hospital. The gomenol caused no febrile reaction. There has been no recurrence of pneumothorax since discharge from the hospital.

Case 2: Mrs. J. Z., age 43 years. White female. Occupation: Housewife. Admitted to the Tuberculosis League Hospital on December 12, 1940, with a diagnosis of spontaneous pneumothorax on the right side.

Twelve years prior to admission, the patient suddenly developed severe dyspnea, associated with pains in the right chest. Following examination, the patient was told that she had a spontaneous pneumothorax; air was removed from the chest and the patient was confined to bed for two weeks. She then returned to her normal activities.

Six years prior to admission, while bending over, she was seized with the same symptoms of pain in her chest and sudden dyspnea. Air again was removed from the chest on several occasions and she was on bed rest for several weeks. She then returned to normal activities and had no recurrence until four weeks prior to her admission to the hospital when she noticed a gradual onset of dyspnea. Spontaneous pneumothorax was again diagnosed and air was removed by the family physician, which alleviated the dyspnea. However, symptoms returned in three days and it was necessary to remove air again. She was then referred to the Tuberculosis League Hospital for treatment.

The patient complained of no symptoms other than shortness of breath. On admission to the hospital the above history was obtained. The patient appeared somewhat dyspneic. The physical examination was essentially negative except for signs of pneumothorax over the right chest and some displacement of the heart to the left. Blood pressure was

136/70. Laboratory findings: Repeated sputum examinations were negative for tubercle bacilli and the sedimentation rate was 16 mm. in 60 minutes; RBC, 4,810,000; WBC, 12,800; Hgb., 94 per cent; Urine, neg.; Kahn, neg. X-ray examination showed a 45 per cent pneumothorax on the right side, with shift of the mediastinum to the left for about one and one-half inches. The lung fields appeared clear.

The day following admission 0.5 of one cc. of gomenol and 10 cc. of saline were injected into the right pleural space. Two more injections of 1 cc. of gomenol and 10 cc. of saline were given, with one day intervals between injections. This created severe pain in the right side and a pleural effusion started to form almost immediately. Due to dyspnea caused by fluid formation, 400 cc. of air were removed on December 13, with a final pressure of −1, −5. On December 20th, 700 cc. of air were removed with a final pressure of +4, −4. Following this, it was not necessary to remove more air. X-ray examination on January 22, 1941, showed the lung to be almost completely re-expanded with some effusion still present in the right pleural space. The patient was then discharged on February 15, 1941. Fluoroscopy at that time showed the lung to be almost completely re-expanded, and the effusion was gradually disappearing. Since discharge from the hospital, the lung has completely re-expanded, the effusion has disappeared, and there has been no recurrence of pneumothorax.

Case 3: Miss F. S., age 21 years. White female. Occupation: Registered nurse. In April, 1945, this patient had a spontaneous pneumothorax on the left side, of about 20 per cent. This was accompanied by slight shortness of breath and occasional pain on deep inspiration. She was in another hospital for three weeks, and returned to work after a month of convalescence. On December 21, 1945, the patient began to experience pain in her chest on deep breathing, and some shortness of breath. This was accompanied by weakness. She lost ten pounds immediately following the onset of these symptoms. X-ray at that time showed a pneumothorax of about 5 per cent over the apex of the left lung. She was then placed on bed rest and by January 1, 1946, the lung appeared to be re-expanded. The patient then returned to work. On February 9, 1946, the patient again experienced the above symptoms, and a pneumothorax of about 20 per cent was discovered on the left side. At that time 1 cc. of gomenol in 5 cc. of novocaine was injected into the left pleural space. This was accompanied by typical severe pain reaction which was controlled by morphine. The patient remained in bed for several weeks; a further x-ray examination on March 22, 1946, showed the left lung to be completely re-expanded, with thickening of the diaphragmatic leaf and a very small amount of effusion at the left base. The patient returned to work on May 1, and has been well since. The lungs were negative for tuberculosis or any other pulmonary infection. The blood count, temperature, and sedimentation rates were always within normal limits.

Case 4: Mr. J. K., age 50 years. White male. Occupation: Materiel man for steel company. Patient gave a history of having been a brick layer, laying fire bricks in a steel mill for 24 years with exposure to dust and sand.

A previous diagnosis of second stage silicosis had been made and the patient was receiving compensation for this disease. Two years prior to admission, the patient suddenly became short of breath and his phys-

ician found a left pneumothorax. He was placed on bed rest for three months, after which time the lung had re-expanded. The patient then returned to ordinary activity and remained well until three weeks prior to admission when he again became quite short of breath after working in the garden. His dyspnea had been getting worse until the time of admission, and x-ray films showed partial collapse of the left lung. Rather sharp pain had been noticed in the left chest. He was admitted to the Tuberculosis League Hospital on April 26, 1946, with the diagnosis of second stage silicosis and left pneumothorax. Physical examination revealed signs of pneumothorax in the left chest, with rales over the right chest anteriorly and posteriorly. Blood pressure was 98/70. Patient was quite short of breath. Laboratory findings: Sputum had been negative for tubercle bacilli for several years, and remained negative while in the hospital; Urine, neg.; RBC, 5,350,000; WBC, 6,250; Hgb., 94 per cent. Sedimentation rate was 10 millimeters in 60 minutes.

X-ray examination on admission showed silicosis, second stage, 20 per cent pneumothorax of the left lung, with adhesions to the first and second interspaces. On the day following admission, 1 cc. of gomenol and 5 cc. of 1 per cent novocaine were injected into the left pleural space. This caused severe pain in the right side, which was easily controlled by morphine. The following day, because of increasing dyspnea, 300 cc. of air were removed from the pleural space. Pleural effusion developed quickly, so that on April 30, a further 300 cc. were removed, and on May 1st, 200 cc. were removed with final pressure of −3, −7. The pleural effusion almost completely filled the pleural space. No further air was removed. The lung began to re-expand rapidly, so that on May 7, there was almost complete re-expansion of the left lung with a pleural effusion extending to the fifth intercostal space anteriorly. He was discharged on May 11, 1946, and an x-ray film one month later showed complete re-expansion of the left lung. There was no effusion present at this time. The patient has remained well since leaving the hospital.

Case 5: Mrs. E. M., age 25 years. White female. Occupation: Housewife. In July 1945, the patient developed a severe pain in the left chest, posteriorly, with some shortness of breath. X-ray films in another hospital showed a spontaneous pneumothorax. She was placed on bed rest for several days, and then returned to normal activity after x-ray films showed the lung had re-expanded. There was no recurrence until May 17, 1946, when she again developed pain and some shortness of breath. A diagnosis of recurrent spontaneous pneumothorax was made. She was admitted to the Tuberculosis League Hospital on May 27, 1946. X-ray examination showed a pneumothorax of 20 per cent on the left side. Laboratory findings: Sedimentation rate was 24 millimeters in 60 min.; Urine, neg.; RBC, 4,720,000; WBC, 16,800; Hgb., 91 per cent.

On the day of admission, 1 cc. of gomenol and 5 cc. of 1 per cent novocaine were inserted in the left pleural space. An intense pleuritis developed with severe pain, but this was controlled by morphine. It was not necessary to withdraw air. A pleural effusion developed rapidly, and the patient had a febrile reaction with a peak of 102 degrees F. for two days. The temperature then returned to normal. On June 3, x-ray examination showed almost complete re-expansion of the left lung with the exception of a small area of pneumothorax over the apex and a pleural effusion extending to the second rib anteriorly. The patient was discharged on June 5. An x-ray film two weeks later showed the lung to be

completely re-expanded with a small amount of effusion at the left base. She has remained well since discharge.

COMMENT

It may be seen that gomenol given intrapleurally sets up an intense sterile pleuritis. However, it is felt that 1 cc. diluted in 5 cc. of 1 per cent solution of novocaine is not sufficient to cause any adverse pleural reaction such as necrosis and perforation. Its use in reexpanding tuberculous lungs for thoracoplasty is probably not advisable.

The only adverse reactions encountered have been pain and fever, the former being easily controlled with morphine, and the latter subsiding of its own accord.

Using the technique described, gomenol has proved to be an excellent drug in the treatment of recurrent spontaneous pneumothorax.

SUMMARY

1. Following a short review of substances used for the production of artificial pleurodesis, a technique is described for the use of gomenol in recurrent spontaneous pneumothorax.

2. Five cases successfully treated by this method are presented.

3. A wider use of gomenol for curing recurrent pneumothorax is suggested.

RESUMEN

1. Después de revisar brevemente las substancias que se emplean para producir la pleurodesia artificial, se describe una técnica para el uso del gomenol en el neumotórax espontáneo recurrente.

2. Se presentan cinco casos tratados satisfactoriamente con este método.

3. Se recomienda el uso más extenso del gomenol para curar el neumotórax recurrente.

REFERENCES

1 Ornstein, G. G., and Lercher, L.: "Spontaneous Pneumothorax in Apparently Healthy Individuals," *Quart. Bull., Sea View Hosp.*, 7:149, 1942.
2 Young, R. A.: "Spontaneous Pneumothorax," *Brit. M. J.*, 2:699, 1936.
3 Brunner, A.: "Die Behandlung des sogenannten idiopathischen spontan Pneumothorax," *Schweiz. med. Wchnschr.*, 71:1210, 1941.
4 Brock, R. C.: "Use of Silver Nitrate in Production of Aseptic Obliterative Pleuritis," *Guy's Hosp. Rep.*, 91:99, 1940.
5 Bethune, N.: "Pleural Poudrage," *J. Thoracic Surg.*, 4:251, 1935.
6 Gowar, F. J. S.: "Artificial Pleurodesis in Relation to Pulmonary Lobectomy," *J. Thoracic Surg.*, 12:234, 1943.
7 Kupka, E., and Wagner, W.: "Intentional Obliteration of Pleural Space by Use of Blood," *Am. Rev. Tuberc.*, 43:291, 1941.
8 Betts, R. H.: "Spontaneous Pneumothorax—Method of Management," *Am. Rev. Tuberc.*, 44:369, 1941.
9 Tyson, M. D., and Crandall, W. B.: "Surgical Treatment of Recurrent Idiopathic Spontaneous Pneumothorax," *J. Thoracic Surg.*, 10:566, 1941.
10 Castex, M. R., and Mazzei, E. S.: "Pneumothorax spontané bénin récidivant," *Arch. méd.-chir. de l'app. resp.*, 12:39, 1937.

The Diagnostic Significance of Clubbed Fingers*

J. K. POPPE, M.D., F.C.C.P.**
Portland, Oregon

Since the association of clubbed fingers with chronic pulmonary disease has been noted from the time of Hippocrates and discussed repeatedly, especially during the past five decades, this report will only include its differential diagnostic possibilities and stress the necessity for chest x-ray examinations and bronchograms in all patients with clubbed fingers. This watch glass deformity of the fingernails, associated with broadening of the terminal phalanges and frequently accompanied by an ossifying periostitis of the long bones, is recognized by several different names. The first description of this deformity of the extremities with chronic empyemas by Hippocrates led to the term Hippocratic fingers. When Marie and Bamberger called attention to the associated thickening and sclerosis of the long bones it became known as the Marie-Bamberger's disease. The term acropathy was applied in 1920 to simplify the nomenclature. Hypertrophic pulmonary osteoarthropathy is probably the most common name in use today for the underlying bony changes. According to Cushing[1] the thickening and sclerosis of the bones is thought to present a later stage of progression of the disease, although Shapiro[2] claims to have noted bony changes precede the clubbing of the nails.

In addition to chronic pleuro-pulmonary diseases, clubbing of the fingers has also been frequently noted in patients with congenital heart disease, subacute bacterial endocarditis, cirrhosis of the liver and chronic intestinal diseases, such as advanced ulcerative colitis, amebic dysentery, and polyposis. It is also noted occasionally in postthyroidectomy myxedema, syphilis and malignancy of the thymus. Occasionally unilateral clubbing has been described in association with aneurism of the aorta and surrounding large vessels on the same side. Loucaides[3] was able to find only 18 such cases in 1932, although a number have been reported since.[4] A familial clubbing of the fingers and toes in the absence of any underlying pathology has also been reported a few times. According to Seaton,[5] until 1938 there were two instances in which the clubbing was present in members of three successive genera-

---

*From the Medical and Surgical Chest Service of Barnes Hospital and Washington University School of Medicine, St. Louis, Missouri.

**Now associated with the University of Oregon Medical School, Mayer Building, Portland, Oregon.

tions and six instances on record of its presence in two successive generations.

The mechanism of development of clubbed fingers and hypertrophic pulmonary osteoarthropathy is still somewhat conjectural and has been discussed at considerable length in a number of other papers listed in the bibliography. The infectious and toxic theories are not well substantiated due to its frequent presence in the absence of any infectious process in a number of cases of congenital heart disease, pulmonary arterio-venous aneurisms, hepatic cirrhosis and some cases of bronchogenic carcinomas without any associated pulmonary infection. The presence of unilateral clubbing associated with aneurism of the aorta and subclavian arteries also suggests a localized interference with the blood supply rather than a generalized toxemia. Compere, Adams and Compere[6] reported no successful attempts in animals at reproduction of hypertrophic pulmonary osteoarthropathy by the injection of toxins or attempted simulation of chronic pleuro-pulmonary diseases until 1935. Mendlowitz has done some very interesting experimental work on this disease during the past few years. By measuring the digital heat elimination and arterial blood pressure he[7] concluded that "the maximum heat elimination and hence the blood flow of the distal phalanges of clubbed fingers secondary to lung or congenital heart disease is usually increased, as well as the digital arterial pressure." A few years later he[8] reproduced a condition similar to congenital heart disease in the dog by anastomosing the left pulmonary artery to the left auricle, thereby greatly reducing the oxygen saturation of the blood by this shunt. Out of four successful experiments only one animal showed hyperthrophic pulmonary osteoarthropathy. Schlicke and Bargen[9] report the frequent occurrence of clubbed fingers in advanced cases of ulcerative colitis, but are unable to give a satisfactory explanation except on the basis of oxygen deficiency due to anemia and circulatory insufficiency in debilitated patients with a chronic disease. The clubbing associated with hepatic cirrhosis could also be explained on a circulatory basis.

The principle purpose of this report is to emphasize the importance and necessity of chest x-ray examinations and bronchograms in every patient in whom clubbed fingers are noted on a routine physical examination. A review of the records of 299 lobectomies for bronchiectasis and chronic lung abscesses at Barnes Hospital in St. Louis revealed complete descriptions of the finger nails in only 129. Of this number 103, or 79 per cent, were noted to have had clubbing of the fingers in varying degrees. Eleven chronic lung abscesses without associated bronchiectasis were present in this group in whom 10, or 90 per cent, were recorded

to have had clubbed fingers. This deformity of the fingers was present in several cases with histories of only six months from the onset of the lung abscess. When the disease was limited to one section of lung which could be completely removed at operation a marked improvement in the finger deformity was observed in some of the younger patients within a year or two following lobectomy.

An effort was then made to compare bronchiectasis with tuberculosis in its effect on the finger nails. At Koch Hospital, the St. Louis City Sanatorium, 276 tuberculous patients were examined. Clubbing of the finger nails was noted in 71, or 25 per cent. Much less cyanosis of the nail beds was observed in the tuberculous patients in whom the marked palor was more suggestive of anemia. The patients in nine different divisions were examined and approximately the same ratio of finger deformity was maintained in both white and colored patients. A higher incidence was present in the males, of whom 192 were examined and 57, or 30 per cent, were found to have finger deformities in comparison with 84 females, of whom only 14, or 16 per cent, had clubbing. The most significant observation was the direct relation of the severity of the disease on the finger nail deformity. The typical watch glass deformity was much more frequent in the acutely ill and terminal patients, even those whose disease was of less than one years' duration, than it was in the chronically ill patients who had spent twenty-five years in and out of the sanatorium. This series seems to compare favorably with the reported incidence of 21 per cent clubbed fingers in 3,551 admissions of tuberculous patients to the Henry Phipps Institute at Philadelphia, although Kline[10] reported only 11 per cent in 100 tuberculous patients picked at random at Ann Arbor, Michigan, with an average duration of symptoms of three and one half years.

The frequency of hypertrophic pulmonary osteoarthropathy in patients with bronchogenic carcinoma is somewhat less marked, apparently being more conspicuous in patients with mixed tumors or malignant adenomas in whom the duration of symptoms is considerably longer. Shapiro[2] reports bony changes in only four per cent of 50 consecutive patients with bronchogenic and metastatic pulmonary carcinoma. The memory of two patients in whom a pulmonary malignancy was first suspected on the basis of clubbed fingers suggests this as one of the valuable early signs. A third year medical student first noticed the finger nail deformity in a young woman (D. M.) of 22 years who brought her baby to the St. Louis Children's Hospital for an examination. He referred the matter to a chest specialist who found an obstruction of the right lower lobe bronchus on bronchography and a mixed tumor of the

bronchus on bronchoscopy. A pneumonectomy was performed with the successful removal of the tumor. This woman had been treated for a chronic, slightly productive cough for several years. A middle aged man was noted to have had clubbing of his fingers for six months before his pulmonary symptoms and findings became sufficient to establish a diagnosis of bronchogenic carcinoma. By this time, the tumor was inoperable, since it recurred within another five months after a total pneumonectomy.

The value of complete chest x-ray examinations and bronchography is further suggested by the case of a 28 year old man which was recently reported by Goldman.[11] Cyanosis and clubbed fingers had been noted in this patient since childhood, at which time a diagnosis of congenital heart disease had been suggested although the signs were not typical. A marked increase in the red blood cells gradually occurred with a count of around eight million, at which time a diagnosis of polycythemia vera was made. Further chest examination revealed a pulmonary lesion in the left lung which proved on subsequent surgical-pathological examination to be a large hemangioma or arterio-venous aneurism. Montaschi[12] reported a congenital lung cyst which was discovered on the basis of clubbed fingers. Since no abnormality was noted on the plain chest film and there were no pulmonary symptoms, a bronchogram was necessary to establish the diagnosis.

SUMMARY

1. Clubbing of the fingers is present in about 80 per cent of severe bronchiectasis and chronic lung abscess patients.

2. Clubbing is also noted in about 25 per cent of cases of pulmonary tuberculosis, particularly in the more advanced stages of the disease.

3. Pulmonary osteoarthropathy may be one of the valuable early signs of bronchogenic carcinoma and mixed tumor of the bronchus.

4. The relative incidence of osteoarthropathy is low in intestinal disease, liver disease, syphilis and the other causes of clubbed fingers in comparison with that found in pleuro-pulmonary and congenital heart disease.

5. All patients with clubbed fingers should have complete chest x-ray examination, including bronchography, unless the diagnosis is obvious on the plain chest film.

RESUMEN

1. Los dedos en clavija ocurren en un 80 por ciento de los enfermos de bronquiectasia avanzada y absceso pulmonar crónico.

2. También se observa este signo en un 25 por ciento de los casos

de tuberculosis pulmonar, particularmente en los períodos más avanzados de la enfermedad.

3. La osteoartropatía pulmonar puede ser uno de los valiosos signos precoces del carcinoma broncógeno y del tumor mixto del bronquio.

4. La relativa frecuencia de osteoartropatía es baja en enfermedades intestinales, hepatopatías, sífilis y en las otras causas de dedos en clavija, comparada con su frecuencia en pleuro-neumopatías y cardiopatías congénitas.

5. Todo enfermo con dedos en clavija debe recibir un completo examen radiográfico del tórax, inclusive de broncografía, a menos que sea evidente el diagnóstico en la simple película radiográfica.

REFERENCES

1 Cushing, E. H.: "Club Fingers and Hypertrophic Pulmonary Osteoarthropathy," *International Clinics*, 2:200-205, June 1937.
2 Shapiro, S.: "Ossifying Periostitis of Bamberger-Marie," *Bull. Hosp. Joint Dis.*, 2:77, April 1941.
3 Loucaides, L.: "Einseitige Trommelschlegelfinger," *Zeitschrift fur Medizin*, 12:724, 1932.
4 Rodgers, R. E.: "Case of Unilateral Clubbing of Fingers," *Brit. M. J.*, 2:439, Sept. 1941.
5 Seaton, D. R.: "Familial Clubbing of Fingers and Toes," *Brit. Med. J.*, 1:614, 1938.
6 Compere, E. J., Adams, W. E., and Compere, C.: "Generalized Hyperthrophic Osteoarthropathy; Experimental and Clinical Study with Report of 2 Cases," *Surg., Gynec. and Obst.*, 61:312, 1935.
7 Mendlowitz, M.: "Same Observations on Clubbed Fingers," *Clin. Sci.*, 3:387, 1938.
8 Mendlowitz, M., and Leslie, A.: "Experimental Simulation in Log of Cyamosis and Hyperthrophic Osteoasthropathy which are Associated with Congenital Heart Disease," *Am. Ht. J.*, 24:141, Aug. 1942.
9 Schlicke, C. P., and Bargen, J. A.: "Clubbed Fingers and Ulcerative Colitis," *Am. J. Digest Dis.*, 7:17, Jan. 1940.
10 Kline, E. M.: "Chronic Hypertrophic Osteoarthropathy," *Univ. Hosp. Bull.*, Ann Arbor, 2:28, May 1936.
11 Goldman, A.: "Cavernous Hemangioma of the Lung: Secondary Polycythemia," *Dis. of Chest*, 9:479, 1943.
12 Montuschi, E.: "Clubbing of Fingers and Toes Associated with Congenital Lung Cyst," *Brit. M. J.*, 1:1310, June 1938.

The Second Stage Thoracoplasty

JAMES W. NIXON, M.D., F.C.C.P., and
JOSEPH T. GOETZ, M.D.
San Antonio, Texas

Many surgeons and patients alike share the belief that the second stage of a paravertebral thoracoplasty, in comparison with the first stage, is a simple procedure. This has not been our observation, especially in so far as the comfort of the patient is concerned. This paper is offered to compare the reactions of the patient after the second stage with those which he experienced after the first stage, and also to present certain data which we have found advantageous in improving the management of the patient before and after the second stage.

Literature concerning the first stage thoracoplasty has been ample and quite understandably has indicated that this first operation, because of its peculiar technical difficulties should be designated as the major bridge to be crossed. Consensus gives proper emphasis to the difficulty in obtaining adequate exposure in the removal of the first rib, a step that has been made immensely more simple however by severing the serratus magnus muscles as suggested by Alexander.[1] We have reason to appreciate the importance of good exposure, since we have twice had the misfortune of injuring the subclavian vein, with the succeeding good fortune, however, of safe repair for each of the injuries. An additional difficulty which we concede as being greater in the first stage operation than in the second concerns the problem of severing the heavy rhomboid muscles. Retraction and adequate exposure are much harder to achieve, and an annoying hemorrhage which accompanies the first stage incision of these muscles is much less noticeable in the second stage procedure.

In reviewing the major difficulties of first stage thoracoplasty and in considering the length of time required for its operation, the surgeon, on first thought, might consider this stage as the one of greater proportion. But what of the patient's clinical course, in comparing the two operations? What are his reactions—and what is his mental attitude? We would rather consider this approach to the subject as the criteria upon which we base our comparison of the two operations instead of the technical surgical difficulties of the operation alone.

Over a period of twenty years, we have had ample opportunity to study the progress of the patient following each stage, and we

have concluded very definitely that in most instances the second stage is not tolerated by the patient as well as the first. Most of our complications have followed the second stage; and of significant importance is the fact that the patient complains of more pain, a situation which requires the use of more sedatives over longer periods of time. In addition, the patient notices a more pronounced loss of function of his arm, since he suffers pain in his attempts to use it. Breathing is more difficult, necessitating for relief frequent use of the oxygen tent and, in some instances, over a longer period of time than is required after the first stage. Coughing, though somewhat reduced by the earlier removal of the top ribs, is decidedly more painful, and is productive only after considerable effort on the part of the patient. Frequently, following the second stage operation, the patient will epitomize the situation by the succinct complaint, "If I could have known this would be so painful, I would never have undertaken it." A complaint rarely heard after the first stage operation.

A further appraisal of the situation reveals that wounds, following the second stage, tend to heal more slowly and have a greater tendency either to become infected or to develop a non-purulent drainage. This handicap is explained by the presence of remaining bits of cat-gut which were not absorbed during the interval between the two stages; and further by the fact that sufficient time usually has not elapsed for the complete re-establishment of the interrupted circulation, with the result that the power of healing and of resistance to infection is lessened.

A final aspect of disadvantage concerning the second stage operation is shown in the loss of vital capacity, a hazard which is much greater than that succeeding the earlier operation, and for the obvious reason that the removal of the long rib sections now deprive a larger area of lung tissue of its previous overlying boney support. A greater degree of lung collapse must inevitably ensue. In the first stage operation, the removal of the first two or three ribs alters only slightly the vital capacity of the patient. We have concluded that we would be within the realm of safety in performing the upper stage of a thoracoplasty upon a patient with a capacity of 1,000 to 1,200 cc., but would certainly be within the realm of danger to perform the second stage with the same vital capacity. In other words, there must be neither too many ribs, nor too great a length of a few ribs removed in this operation. If a greater degree of relaxation of the lung should be required, it is far wiser to do three or four operations rather than to harass and endanger the patient with the excess difficulties that attend the crowding of the work into two stages.

Concerning management, we have found that the length of

time allowed to elapse between the first and the second stage operations is a very important factor. Formerly, it has been our custom to perform the second stage at the end of ten or twelve days. Later we extended this period to fifteen days, but more recently we have insisted that eighteen to twenty-one days must intervene before the second stage is undertaken. This increased interval makes it possible for the patient to come to the second stage in far better condition. During the last week of this intervening period the patient's appetite improves, his cough diminishes, and the freedom from pain allows him to have his much needed sleep and rest. Furthermore, we are able to have him out of bed for a few days, between stages, which circumstance has an important influence on the circulation and the general well being of the patient as well as giving an impetus to wound healing.[5] In addition, the patient's morale invariably improves when he is permitted out-of-bed privileges. Ochsner[2] has pointed out the importance of ambulation, both pre-operatively and post-operatively, for this type of patient.

During the interim other advantageous results can be accomplished in preparing the patient and in lessening the danger for him. We advocate early feeding, and in fact begin feeding the patient almost within twenty-four hours after the first operation. Since hypoprotonemia is frequently associated with chronic lung infections, we believe that special emphasis should be placed on a high protein diet. And because of the importance of vitamin C in wound healing, we suggest a daily dosage of 100 milligrams of ascorbic acid to be given the patient for a week prior to each operation, but this is of especial importance before the second stage.

We have found that there is rarely a need for a blood transfusion during the first two or three days of the interim period, since we routinely transfuse the first-stage patient on the operating table, giving 500 to 1,000 cc. of whole blood. Some four or five days after the operation, we give whole blood transfusions until the red blood cell count is above 4,000,000, and the hemoglobin above 70 per cent, thus assuring the patient a greater margin of safety.

Another routine which we follow is that of administering penicillin to all of our patients for two days prior to the operation, and for three days succeeding the operation also, the amount to be 20,000 units every three hours. For the comfort of the patient who may complain of disturbed sleep by the administration of dosage during the night, we have discontinued the penicillin during those hours; unless, of course, infection has already developed. In our opinion, the importance of rest in the prevention of in-

fection supersedes the advantage which might be gained by routine dosage of penicillin throughout the night.

Before closing, we should like to offer a suggestion concerning the choice of anaesthetic to be used since we believe this factor to be a highly important one in second stage thoracoplasty. For the previous operation we have found it advantageous to employ intratracheal anaesthesia, because of the difficulty encountered in obtaining relaxation of the heavy muscles attached to the scapula. However, for the second stage, we have abandoned the use of this type of anaesthesia, since fewer muscles are encountered. Instead, during the past three years we have employed sodium pentothal intravenously, supplemented with local anaesthesia of one per cent novocaine. The sodium pentothal brings about a quiet induction, and the patient, released from fear, is quickly brought to the stage of surgical anaesthesia. We find an advantage in supplementing the main anesthetic with novocaine, since the entire operating procedure can be carried out with a minimum amount of pentothal, thus lessening the likelihood of respiratory depression, a condition which would otherwise be an objectionable feature to this method of anaesthesia. In our experience, we have had only one patient who had respiratory difficulties, which trouble we attributed to the fact that three grains of nembutal had been given the patient three hours prior to the operation. As is well known, this sedative is definitely contraindicated when sodium pentothal is used. Another advantage which ensues when this method of anaesthesia is used, concerns the amount of secretion which may accumulate in the trachea and bronchi. Not only is the amount less following the operation, but the small amount which does accumulate is coughed up easily by the patient because of quickly returning reflexes. Coughing is made easier by the reason of the fact that the paravertebral anaesthesia block lessens the pain and does not cause the patient to limit his coughing excursion. Gurd[4] advocates spinal rather than general anaesthesia for the second stage and for the same reason that we have abandoned the general anaesthesia. Because the results we have experienced in our method have been so satisfactory, we feel justified in recommending it.

SUMMARY

The clinical reaction and the mental attitude of the patient, following the first and second stage of a paravertebral thoracoplasty, are contrasted, correcting the often accepted belief that the second stage is of less importance to the patient than the first.

From experience of twenty years it is concluded that the second stage is not tolerated as well as the first. There is a greater inci-

dence of complications. Increased pain on coughing and on use of the arm requires more sedation. Respiration is more difficult, requiring an oxygen tent for a longer period. The wound heals more slowly and exhibits a greater tendency to either become infected, or to develop a non-purulent drainage, due to remaining bits of catgut not absorbed in the interval between the two stages and, because of incomplete re-establishment of circulation. Finally, the loss of vital capacity is greater.

Measures adopted to lessen the gravity of the second stage thoracoplasty consist of the following:

1. Increasing the interval between stages from ten or twelve days to eighteen or twenty-one days. This time shows improvement in appetite, diminished cough and allows much needed sleep and rest. Ambulation for a few days has an important influence on the circulation, general well-being, and, morale of the patient.
2. Feeding early, with special emphasis given to a diet high in protein content. Blood transfusions are added as needed and vitamin C is administered.
3. Combatting infection by use of penicillin.
4. Choosing an anesthetic which lessens complications. Intravenous sodium pentothal supplemented by paravertebral block with one per cent novacaine is recommended. A quiet induction, quick return of cough reflexes, lessened secretions, and decreased pain on postoperative coughing are advantages of this method.

RESUMEN

Se comparan la reacción clínica y la actitud mental del enfermo siguientes a la primera y segunda etapas de una toracoplastia paravertebral, y se corrige la creencia, frecuentemente aceptada, de que la segunda etapa es, para el paciente, de menor importancia que la primera.

A base de la experiencia de veinte años se concluye que no se tolera la segunda etapa tan bien como la primera. Sobrevienen con mayor frecuencia las complicaciones. Se siente más dolor al toser y al mover el brazo, lo que requiere más sedantes. Es más difícil la respiración y se necesita la tienda oxígena por un período más largo. Se cicatriza más despacio la herida y manifiesta mayor tendencia a infectarse o a producir un drenaje no purulento, causado por los pedacitos de catgut que no se han absorbido en el intervalo entre las dos etapas, y debido a la incompleta restauración de la circulación. Finalmente, es mayor la pérdida en la capacidad vital.

Han sido adoptadas las siguientes medidas para disminuir la gravedad de la segunda etapa:

1. Prolongar el intervalo entre las etapas de diez o doce días a dieciocho o veintiún días. Este período permite que mejore el apetito y disminuya la tos, y proporciona sueño y descanso que son muy necesarios. El levantarse por unos cuantos días ejerce una importante influencia sobre la circulación, el bienestar general y el estado de ánimo del paciente.
2. Alimentar al enfermo pronto, recalcando especialmente la importancia de una dieta rica en su contenido de proteínas. Se dan transfusiones de sangre cuando son necesarias y se administra vitamina C.
3. Combatir infección mediante el uso de penicilina.
4. Escoger un anestésico que disminuya las complicaciones. Se recomienda el pentatol sódico por la vía intravenosa, reforzado por bloqueo paravertebral con novocaína al uno por ciento. Las ventajas de este método son: tranquila inducción, rápida reaparición de los reflejos de la tos, disminución de secreciones y menor dolor con la tos postoperatoria.

REFERENCES

1 Alexander, J.: "Special Consideration Relating to Surgical Closure of Large Upper Lobe Tuberculous Cavities," *J. Thorac. Surg.*, 2:1, 1932.
2 Ochsner, A.: "Preoperative and Postoperative Treatment of Thoracic Surgical Cases," *S. Clin. North America*, 25:1106, 1945.
3 White, W. L. et al: Cited by Ochsner.[2]
4 Gurd, of Montreal: Personal communication.
5 Nixon, J. W.: "Early Postoperative Walking," *S. Med. J.*, 37:682, 1944.

Observations on Cystic and Bullous Emphysema of the Lungs*
A Study of 100 Cases

EPHRAIM KOROL, M.D.
Halloran Veterans Hospital
Staten Island, New York

Classification

The two forms of emphysema are distinguished from each other largely by the size of the emphysema cavities and by the association with other pulmonary diseases.

In cystic emphysema the cavities are very large, often simulating pneumothorax on the roentgenogram, and there is generally no accompanying infectious process in the lungs. The cysts involve the upper and mediastinal parts of the lungs, regions not very active in air exchange. Symptoms are few and these cases are generally discovered accidentally.

In bullous emphysema the cavities are smaller and more numerous, and in this condition there is invariably an associated chronic lung disease such as asthmatic bronchitis, bronchiectasis, tuberculosis, or silicosis. The more active basal portions of the lungs are involved, and the symptoms are pronounced.

The cystic cavities appear early in life and increase in size gradually with the growth of the chest, without encroaching on the adjacent lung. This is one reason the condition may develop to a spectacular degree before it is discovered clinically.

Bullous emphysema is a disease of adults, and the cavities develop and progress at the expense of the neighboring lung lobules.

Pathogenesis

Emphysema is conceived as an atrophy of the lung caused by the effect of an inadequate blood supply to an organ functioning, at times, under great stress.

The bronchial artery, the nutritional vessel of the lung, is the smallest of the visceral arteries. The lung is more vulnerable to vascular accidents such as embolism, gangrene, infarction, and hemorrhage, than any other organ, and the degenerative process of emphysema is attributable to the same vascular factor.

Postmortem the anemia of the emphysematous lung is striking,

*Presented at the Twelfth Annual Meeting, American College of Chest Physicians, San Francisco, California, June 28, 1946.

and histologically there is an obvious disappearance of the capillaries. Roentgenologically, there is increased transparency of the lung caused by a loss of the vascular markings; the larger pulmonary trunks, however, undergo enlargement and densification.

All the lung elements and the alveoli are complete in childhood. With the growth of the chest the lung undergoes enlargement chiefly by stretching and confluence of alveoli. The blood supply is adequate for the nutrition of the bronchi, but in the periphery of the lung atrophic changes appear early in life. By middle life hardly an individual escapes the degenerative change in the lungs.

In asthma and in other states of bronchial spasm and bronchial stneosis the progress of emphysema is enhanced by the overinflation and stretching of the lung. With the relief of the bronchial obstruction, the lung will not often return to the original state, but will remain emphysematous, due to the inadequate vascularization.

Review of 100 Cases

One hundred cases of emphysema have been studied with particular reference to the origin and course of the disease. The subjects are veterans of the Spanish American War and veterans of World War I. All of the cases have been under observation for several years, some of them for 20 years and longer. Thirty cases were followed to postmortem examination.

In several cases the lung changes began and developed while under observation; in the majority, emphysema was present when the patients first presented themselves; in most cases there was a tendency to steady, though slow, unfavorable progression. In 3 cases temporary improvement was registered on the series of roentgenograms.

Our data indicate that cystic emphysema is not usually a congenital condition but is acquired, generally, early in life. In many cases the roentgenographic evidence of the disease manifests itself after military age is reached.

In case XC-285 304, the histological sections of the lungs show the text-book picture of congenital lung cyst. However, the available clinical and x-ray records show conclusively that the condition developed since the man's military service.

Cystic emphysema is often associated with the ordinary diffuse form of vesicular emphysema, the air cysts representing an exaggeration of the atrophic changes in certain areas of the lungs. Common to the two forms of emphysema are the vascular changes, namely destruction and disappearance of the capillaries and atherosclerotic and endarteritic changes in the large pulmonary vessels.

An impressive number of these cases, 28 in number, had advanced systemic arteriosclerosis and hypertension. This finding is in agreement with the vascular theory of emphysema: The blood supply to the lung is further impaired by the general vascular inadequacy.

There is a surprisingly large number of negroes in the group, 16 cases. The explanation would lie in the greater incidence of vascular disease among negroes.

Syphilis occurred in 15 of the 100 cases, and is considered an important etiologic factor, prone as it is to attack the vascular system. Tuberculosis, 19 cases, and silicosis, 9 cases, are other important factors.

The diagnosis of pulmonary syphilis was entertained clinically in 4 cases and was suspected in several others. In 3 cases the pathologist described vascular changes in the lungs which are considered indicative of syphilis.

In the chronic forms of tuberculosis there was overinflation and stretching of the uninvolved portions of the lungs caused by mechanical factors and altered space relations between the contracting lung and the non-yielding bony thorax. The loss of abdominal fat and of muscle tone in the abdominal muscles causes a descent of the diaphragm with stretching of the basal portions of the lungs. The decrease in the size of the heart has a similar stretching effect on the lungs.

In the more acute cases of tuberculosis bronchostenotic lesions were operative.

In four cases there was compression of a main bronchus by enlarged lymph nodes.

In the cases with silicosis periarteritic fibrosis was conspicuous. Otherwise the same factors were operative as in chronic tuberculosis.

Allergic bronchitis and asthma was present in 23 cases and produced progressive emphysema. The resultant air cysts appearing in the series of roentgenograms differ in on way from those developing in cases without bronchial spasm.

A most interesting and unexpected finding was the development of bronchial carcinoma in seven of the cases of cystic emphysema. No bronchial carcinoma has as yet developed in any of the cases of bullous emphysema in this group.

SUMMARY

The author analyzes 100 cases of advanced Bullous and Cystic Pulmonary Emphysema.

In Bullous Emphysema there is a causative or associated lung infection and there are disabling symptoms. Cystic Emphysema develops early in life and progresses without bronchial infection

and without bronchial spasm, and gives few subjective complaints.

In most cases of Emphysema there are signs of vascular degeneration: disappearance of the smaller pulmonary radicles and arteriosclerotic or endarteritic changes in the larger pulmonary arteries.

A discussion of the vascular factor in the pathogenesis of emphysema is included.

RESUMEN

Analiza el autor 100 casos avanzados de Enfisema Pulmonar Vejigoso y Quístico.

En el Enfisema Vejigoso existe una infección pulmonar causal o asociada, y ocurren síntomas incapacitantes. El Enfisema Quístico aparece a una edad temprana, progresa sin infección o espasmo bronquiales y ocasiona pocos síntomas subjetivos.

En la mayor parte de los casos de Enfisema existen síntomas de degeneración vascular, a saber: desaparición de las más pequeñas radículas pulmonares y alteraciones arterioescleróticas o endarteríticas en las más grandes arterias pulmonares.

Se incluye una discusión del factor vascular en la patogenia del enfisema.

Treatment of Complications Arising in the Course of Pneumothorax Therapy of Pulmonary Tuberculosis*

MORTON R. GIBBONS JR., Commander (MC) USNR
Corona, California

Introduction

The tuberculosis unit of United States Naval Hospital at Corona, California, was commissioned in July of 1943. The purpose of the unit was to care for patients suffering from established active pulmonary tuberculosis. By reason of the usual rotation of medical officers, the staff of this unit has experienced continuous change and therefore the individual care of various patients has shown a variety of methods and concepts, although the policies of the institution have, in general, remained the same.

The statistical data concerning U. S. Naval Hospital, Corona, California, Tuberculosis Unit from opening date, July, 1943 to May 1, 1946 is as follows:

Total admissions	2,838
Total discharges	1,713
Readmissions	18
Patients remaining	1,025
Deaths	82

In one significant characteristic, the care of the patients in a Naval hospital varies from the usual course of treatment of tuberculosis. The average stay of the patient in the hospital is about 8 months only. The Navy does not care for these patients until their ultimate recovery or other termination of treatment except in the case of officers who have been retired by reason of their disease or in the case of enlisted men belonging to Fleet Reserve. Following this policy, it has been the aim of the hospital to attempt to control the activity of a tuberculous infection by such means as may be indicated and then to transfer the patient to a Veterans' Administration hospital for convalescent or further care. It will be evident from this explanation that the staff of the hospital has not been able to follow its cases for a determination of the end results of the treatment instituted.

---

*Presented at the Twelfth Annual Meeting, American College of Chest Physicians, San Francisco, California, June 29, 1946.

**From the Tuberculosis Service, United States Naval Hospital, Corona, California. This paper reflects the views of the author and is not to be considered as representing the policies of the Medical Department, U. S. Navy.

The purpose of this paper is to set forth the methods which have been evolved in the organization above described for the treatment of complications which have developed during the course of pneumothorax therapy..

Bedrest has been the essential factor in all treatment and is instituted and associated with all other methods of treatment discussed.

Pneumothorax has been considered routinely in all cases of proved active tuberculosis except those which show a fairly stable minimal involvement. The majority of minimal cases now undergoing pneumothorax therapy in the unit had their procedure instituted elsewhere. All suitable cases should have pneumothorax attempted, and further procedures should be carried out only if it is not successful. Individuals demonstrating dense pneumonic tuberculosis are considered unsuitable for pneumothorax and there is a general disinclination to its institution during an acute, toxic febrile reaction.

Temporary interruption of the phrenic nerve has been practiced extensively and has been used alone, with pneumothorax, or with pneumoperitoneum and other procedures. We have experienced gratifying results in its use in many cases under varying conditions.

Pneumoperitoneum, in connection with paralysis of one wing of the diaphragm, has had a rather more extensive use than at some other institutions. The results of this procedure have likewise been gratifying, particularly in those cases where no other form of collapse therapy could be adopted.

In a paper having the scope of this, it is not possible to discuss the more radical surgical procedures, and therefore, it is proposed to limit discussion of complications to those experienced in the above procedures.

The complications of pneumothorax are divided indefinitely into two groups: immediate (i.e., associated with the acts of instituting or refilling pneumothorax) and, late.

Classification of Complications of Pneumothorax Treatment

I Incident to the performance of pneumothorax.
- a. Early
  1. "Pleural shock"
  2. Pulmonary embolism
  3. "Spontaneous" pneumothorax
  4. Chest wall injury—hemorrhage
  5. Subcutaneous emphysema.
- b. Late
  1. Pleural effusion

2. Spontaneous rupture of pleural adhesion
3. Gross interference with vital capacity.

II Incident to intrathoracic changes caused by pulmonary infection.

a. Tuberculous pleurisy and empyema
b. Atelectasis
c. Bronchopleural fistula and secondary infection
d. Chest wall abcess.

III Conditions interfering with adequate pneumothorax.

a. Pleural adhesions
b. Tension cavity.

Prevention is the best treatment for immediate complications. The patient is prepared for the initial pneumothorax, the procedure and the results desired being explained. Sedation, pentobarbital solium grs. 1½, is usually administered to initial cases. The anesthetic is 1 per cent procaine and it is injected through a 1½ inch 22 gauge infiltration needle after the skin has been cleansed with tincture of merthiolate. The procedure has been to raise a wheal by intradermal injection in skin stretched between the thumb and forefinger and then to follow through the same wheal until the parietal pleura is reached, this structure being then anesthetized by further injection of procaine. The site of election is the 5th intercostal space in the anterior axillary line unless disease immediately underlying this area contra-indicates. One should go above rather than below a rib. Following anesthesia, the infiltration needle is withdrawn and an 18 gauge 1½ inch needle, attached by tubing to the manometer of the pneumothorax machine, is inserted into the pleural space. It is generally preferred that a short bevel needle be used for initial pneumothoraces, and when such needle is used, it is helpful to make a small incision in the skin with a scalpel, or, better, to puncture the skin with a sharp bevel 18 gauge needle prior to the insertion of the pneumothorax needle. Before the administration of air, it is absolutely essential that good negative manometric readings be obtained, usually ranging between −14 to −8 cm. H_2O on inspiration and −8 to −4 cm. of H_2O on expiration. At the time of initial administration, and following recording of the manometric reading, 50 cc. of air is introduced slowly followed by a second reading of the manometer. From the change in pressure brought about by the 50 cc. of air, an estimate of the patency of the pleural space and the amount that can be safely given can be made. It has been the practice to administer 300 cc. at the initial introduction of air and to increase this by 100 cc. every 4th day but rarely exceeding

500 cc. at any one administration. The patient remains flat in bed for the first 24 hours following the initial procedure. He then is routinely fluoroscoped and subsequent course may be determined by the picture visualized. At the unit there has been no recorded case of so-called "pleural shock," which is believed now to be actually air embolism. Aside from an occasional case of syncope associated with vaso-motor disturbances, similar in all ways to surgical shock, there have been no untoward reactions except for one case which displayed a bizarre form of central nervous system stimulation which was diagnosed as being of psychogenic origin.

Inasmuch as fluoroscopy 24 hours following an initial pneumothorax not infrequently shows the presence of what is apparently a greater amount of air than introduced, the belief has been advanced that a small, self-limiting traumatic pneumothorax, presumably from inadvertant puncture of parenchyma may be a concurrent phenomenon of most initial pneumothoraces. If this is so, it has been our experience that such extrusions of air from the lung into the pleural space are always self-limiting and do not produce fistulae, empyemata or any unwarranted degree of pleural effusion. Neither do they result in massive collapse or subcutaneous emphysema and they are usually unrecognized by the patient. The treatment of this condition is expectant only. The patient is fluoroscoped regularly and more air introduced only when the lung has re-expanded to an undesirable volume.

All patients undergoing collapse therapy should be fluoroscoped at least twice weekly, immediately preceding the regular pneumothorax refills. After a pneumothorax has been established satisfactorily, refills are administered only on the basis of the appearance of the collapse under the fluoroscope. About 50 per cent collapse is maintained, allowing, if possible, a selective collapse of the diseased parts with relative re-expansion of healthy lung. It is recommended that bronchoscopy be performed on every case in which collapse therapy is indicated. The value of this procedure in diagnosis and the determination of the condition of the air passages definitely outweighs any possible danger. The presence of endotracheal or bronchial disease considerably reduces the possibility of an effective pneumothorax and predisposes to atelectasis and check-valve cavity formation.

Effusion

A clear, sterile fluid containing no bacteria frequently accompanies pneumothorax, and is usually confined to the costophrenic angle. In the absence of symptoms its presence can be ignored since it has no bearing on the success of the treatment.

Empyema

Empyema is any collection of fluid in the pleural space containing bacteria. Etiological classification is as follows:

1. Tuberculosis of the pleura.
   a. Acute
   b. Chronic
2. Spontaneous pneumothorax.
3. Spontaneous rupture of pleural adhesions.
4. Bronchopleural fistula.
5. Postoperative.

The above conditions are interlocked, they may occur simultaneously or as the result of one another. Therefore, a discussion of the subject as a whole rather than individual consideration of these conditions seems indicated.

A second and third type of classification of empyema might be based on the types of fluid present and the degree of control of the underlying pulmonary tuberculosis and a fourth might be based on the reaction of the patient to the presence of the empyema, regardless of the cause of the condition.

The commonest cause of empyema is tuberculosis of the pleura, which may become associated with pneumothorax collapse at any time during the course of the therapy. The onset may be hidden and asymptomatic and the presence of the fluid discovered by radiography. Again there may be a systemic reaction of fairly sudden onset of chest pain, fever, and elevation of sedimentation rate. In every case a sample of the fluid should be removed for analysis, and intrapleural manometric readings should be taken. In tuberculous empyema, not complicated by an interruption of the continuity of the visceral pleura, there will be no marked immediate change in the configuration or collapse of the underlying lung and no marked shift to a positive pressure accompanying the development of the empyema. This will serve as a diagnostic sign by which the physician may differentiate this condition from those in which there is actual or potential secondary infection.

The treatment of this condition depends upon several factors such as the nature of the fluid, the type of collapse under the fluid and the degree of control of the disease in the underlying lung. No attempt is at first made to evacuate the fluid material. Pneumothorax is continued for as long as possible and according to the usual manometric readings. Many of these cases show a spontaneous resorption of the empyema without adverse effect upon the pneumothorax or the underlying lung. If, as occasionally occurs, the pleural space becomes completely obliterated by fluid,

and if the underlying pulmonary disease is well under control, the pneumothorax may be abandoned with the hope that the gradual expansion of the underlying lung in the presence of the slow resorption of the empyema will prove beneficial to the patient. If, however, the underlying lung is not under control and particularly if healthy lung is being jeopardized by contra-selective collapse, it would seem wiser to evacuate the fluid, allow the lung to re-expand, and consider thoracoplasty or other more permanent surgical procedure. If the onset of this type of empyema is acute, it will be found that the severer toxic symptoms frequently disappear within a week or so and the patient rests comfortably without inconvenience from the fluid in the chest. There is not infrequently found to be a marked subjective improvement in the patient following the development of fluid in the pleural space, particularly when that fluid almost or completely obliterates the space and the underlying lung is well controlled. Thick, purulent empyemas are more dangerous than serous or seropurulent types, and one is more inclined to attempt reexpansion and obliteration of the pleural space with later thoracoplasty when pus is present. To avoid the development of a pleuro-cutaneous sinus or chest wall abcess, it is advised that needles be inserted into the pleural space as infrequently as possible.

Spontaneous Pneumothorax

In spontaneous pneumothorax, we are faced with a condition which may bring about one or all of several undesirable complications. This may be small, self-limiting and relatively asymptomatic, but empyema, atelectasis, broncho-pleural fistula, subcutaneous emphysema and gross interference with vital capacity may result. The same holds true in the case of a spontaneous separation of pleural adhesions, though this latter condition can occur without the escape of air into the pleural cavity. The treatment should be expectant, and depends on the reaction of the patient and how well the condition cares for itself. Where there has been rupture of the visceral pleura, penicillin is administered immediately. The dosage is 20,000 units intramuscularly every 3 hours, and 50,000 units intrapleurally every day, until all chance or evidence of secondary infection has passed. Where the escape of air into the pleural space is sufficiently great to embarrass respiration, it is necessary to remove air. This is done preferably by reverse pneumothorax, and the intrapleural pressure should not be allowed to fall below atmospheric pressure. The increased pressure in the pleural space is an aid to the sealing off of the leakage from the lung, and is only reduced when absolutely necessary. If a large broncho-pleural fistula permits too much air to

escape, continuous decompression must be established. The need for this depends on interference with vital capacity or the presence of extending subcutaneous emphysema. Obviously, it is not necessary to refill the pneumothorax space unless the fistula becomes closed.

Broncho-pleural Fistula

The diagnosis of broncho-pleural fistula is established by the instillation of 1 per cent aqueous solution of methylene bile into the pleural space and its subsequent recovery in the sputum. Such fistulae can close spontaneously, and should be given an opportunity to do this before surgical intervention is tried. The patient should lie on the affected side.

We have had no experience with attempts to obliterate the pleural space by oxygen lavage, oleothorax or detergent solutions. The same considerations as those given above apply to empyema following surgical invasion of the pleural space.

Atelectasis

Atelectasis is a frequent companion to pneumothorax. It is probably present in some degree in almost all established pneumothoraces. The causes are either an obstruction of the air passages or the establishment of too great a collapse by external pressure. Obstruction of the passages may result from endobronchial disease, mechanical obstruction, or alteration and angulation of the bronchi as a result of alteration of intrathoracic anatomical relationships by pneumothorax and pleural adhesions. The condition might be anticipated and avoided by bronchoscopy prior to the institution of pneumothorax in some cases.

If cavitation occurs in an atelectatic area, it is quite likely to be of the type called "blocked," "tension" or "check-valve." An early estimate of the possibility of atelectasis and concurrent "blocked" cavity in the upper lobes may be made from the type of collapse following the establishment of an initial therapeutic pneumothorax. If the apex, when collapsing, falls away from the mediastinum, and collapses toward the hilus, atelectasis and "blocked cavity" are not likely to occur. If, on the other hand, the apex is adherant medially to the mediastinum and, on collapse, falls away from the lateral pleura towards the mediastinum, a mechanical angulation of the upper lobe bronchus is possible and the conditions under discussion can be produced.

The presence of extensive atelectasis is an indication for allowing partial or complete reexpansion of a collapsed lung. It is suggested that during this procedure, the operator may increase and decrease the collapse alternately for a few days to try to

"juggle" the lung to release any mechanical obstruction but. it should be remembered that loss of use of a portion of the lung is occasionally the price paid for the healing of a tuberculous process therein, and it may be better to lose the use of a lobe if such loss results in the control of the disease.

"Blocked Cavity"

As noted above, "blocked cavity," may be associated with atelectasis, and in such case, the method of handling may be the same. It will occasionally be noted that cavities existing prior to pneumothorax, which take on the attributes of a "blocked cavity" under collapse therapy, may actually disappear if the lung is allowed to reexpand. Such cavities frequently increase in size when undergoing attempted collapse, and various methods have been tried in an effort to eliminate them. Restraining adhesions should be severed, if possible. Where the pull caused by inoperable adhesions at the apex and over the diaphragm maintains tension which interferes with adequate lateral collapse, phrenic crush may release such tension to a degree where the pneumothorax can produce an effective result. Failing the lesser measures, it has been the policy to allow reexpansion of the collapsed lung with a view to the performance of other surgical procedures, usually thoracoplasty.

Pleural Adhesions

Pleural adhesions of varying size and location have been a complicating factor in something over 50 per cent of the established pneumothoraces. In the hands of the various members of the surgical staff, the operation of closed intrapleural pneumonolysis has given excellent results, and in almost every case where adhesions have been thought to interfere with collapse a thoracoscopy at least has been performed. We have also used temporary phrenic paralysis to release tension of adhesions with good results where pneumonolysis could not induce adequate collapse. The presence of pleural adhesions need not necessarily prevent an adequate collapse, as demonstrated by the disappearance of cavitation and the conversion of the sputum. If inoperable pleural adhesions definitely prevent adequate collapse, and interdict effective results from pneumothorax therapy, the lung should be reexpanded and more severe surgical procedures considered.

The accompanying tables indicate the occurence of complications of collapse therapy. Table 1 indicates the various procedures associated with the institution and maintenance of collapse therapy over a 3 year period. Table 2 indicates the complications at present existing in patients now under treatment.

TABLE 1

Total number of procedures associated with the institution and maintenance of collapse therapy at U. S. Naval Hospital, Corona, Tuberculosis division, from open date, July 13, 1943 to May 1, 1946.

Pneumothorax	34,464
Pneumoperitoneum	482
Aspirations	534
Bronchoscopy	212
Temporary Phrenic Nerve Interruption	233
Closed intrapleural pneumonolysis	764
Thoracoplasty (all types and stages)	259
Miscellaneous procedures	97

TABLE 2

		Minimal	*Moderately Advanced*	*Far Advanced*
Total number of Patients	582	106 (18.2%)	295 (50.7%)	181 (31.1%)
Pneumothorax	351 (60.2%)	13 (12.2%)	192 (65.1%)	146 (80.7%)
Empyema (all types)	98 (27.9%)	2 (15.5%)	43 (22.4%)	40 (27.5%)
Empyema, purulent	13 (3.7%)	0	3 (1.5%)	10 (6.8%)
Spontaneous Pnx.	15 (4.2%)	0	5 (2.6%)	10 (6.8%)
Adhesions	200 (56.9%)	5 (38.4%)	100 (52.6%)	94 (64.4%)
Blocked Cavity	26 (7.4%)	0	10 (5.2%)	16 (10.9%)

TABLE 3

Table 3 shows the pneumothoraces, complications and more common surgical procedures performed on 1,024 consecutive patients whose histories were presented to the Board of Medical Survey for transfer, discharge, or retirement.

	Total Patients	1,024	
Pneumothorax	663 (67.7%)	Pnx. abandoned	54 (8.1%)
Empyema (all types)	78 (10.2%)	Pneumonolysis done	273 (41.2%) (76.0%)
Empyema, purulent	10 (1.5%)	Aspirations	30 (4.5%)
Spontaneous Pnx.	19 (2.9%)	Phrenic Crush	54 (8.1%)
Adhesions	357 (53.8%)	Thoracoplasty	29 (2.8%)

SUMMARY

This paper sets forth the statistics of a large U. S. Naval Hospital Tuberculosis Unit with special attention to the occurrence and treatment of complications associated with Pneumothorax Therapy. Such complications are classified as those resulting from the

performance of pneumothorax and those resulting from coincident intrathoracic changes.

An outline of suggested pneumothorax technique is presented with the suggestion that proper procedure will eliminate complications. In complications resulting from intrathoracic changes, it is suggested that the patient suffer a minimum of interference; that the condition of the patient and his response to his disease rather than the mere presence of fluid, adhesion or other complication be the determining factor for the institution of surgical procedures. The various complications are discussed as regards treatment, particularly with the aim of their elimination without the forced abandonment of the pneumothorax and the performance of more permanent surgical procedures.

Finally, tables of the collapse procedures instituted, the complications arising and the measures used to combat these in 1,048 consecutive cases are presented.

The author gratefully acknowledges the valued assistance of E. G. Brian, Capt. (MC) USN (Ret.), S. T. Allison, Capt. (MC) USNR, Norma C. Furtos, Lt. Comdr. (MC) (W) USNR, and James H. McCorkle, Lt. Comdr. (MC) USNR.

RESUMEN

Se presentan en este trabajo los datos estadísticos de las Salas para Tuberculosos de un gran Hospital Naval de los Estados Unidos, prestándole especial atención a la aparición y el tratamiento de complicaciones asociadas con la Terapia Neumotorácica. Se clasifican esas complicaciones en las que son ocasionadas por la ejecución del neumotórax y las que se deben a cambios intratorácicos coincidentes.

Se presenta un bosquejo de una técnica que se recomienda para la administración del neumotórax y se indica que el empleo de un procedimiento correcto eliminaría las complicaciones. En las complicaciones ocasionadas por alteraciones intratorácicas se sugiere que se someta el enfermo a un mínimo de intervención; que la condición del paciente y su reacción a la enfermedad, más bien que la mera presencia de líquido, adherencias u otra complicación, sea el factor determinante para la aplicación de procedimientos quirúrgicos. Se discuten las diferentes complicaciones en cuanto a su tratamiento, particularmente con el objeto de eliminarlas sin el abandono forzado del neumotórax y la ejecución de procedimientos quirúrgicos más permanentes.

Finalmente, se presenta una tabla de los procedimientos de colapso que se aplicaron, las complicaciones que sobrevinieron y las medidas que se emplearon para combatirlas en 1,048 casos consecutivos.

Discussion

JOHN ROBERTS PHILLIPS, M.D., F.C.C.P.
Houston, Texas

It is good to discuss an old procedure. Pneumothorax, a relatively simple surgical procedure, has controlled, by itself or with other types of surgery, more tuberculosis than any other operation.

As a whole, the complications from it are relatively few, rather the complications are from the disease. About fifty per cent have adhesions which require severance, sometimes requiring more than one attack in order to get sufficient separation, so that cavity closure will result. If adhesions prevent closure of cavities, a phrenic crush may cause enough relaxation to effect closure. The success of pneumothorax by itself, or with other procedures, occurs in a high percentage of cases, as shown by the low incidence of abandoment in Commander Gibbons' series. Once it is seen that cavities will not close, it is useless to compress good lung and allow adhered diseased areas unaffected to remain expanded. Consequently, the pneumothorax should be abandoned and other collapse measures or resection of the diseased portion resorted to

A complication that might be serious is tension in a spontaneous pneumothorax. It can readily be relieved by putting a needle in the chest and connecting it by tubing under water. Fluid in the chest may follow, which usually is not serious.

Many cases of tension pneumothorax are seen particularly in civilian and war injuries. It's management is easy once the diagnosis is made. A careful study and selection of procedure in an individual case is essential for best results. Complications will be less and less when this is adhered to.

Discussion

JOSEPH ROSENBLATT, M.D., F.C.C.P.
Los Angeles, California

With the exception of cases of gross negligence when air is introduced into the chest in the absence of satisfactory manometric readings, immediate complications are the result of injury to the lung by the pneumothorax needle. Since it is readily admitted that the lung is frequently punctured at the initial operation, and this is inevitable in cases where there is no free pleural space at the site of the puncture, it is reasonable to assume that the smaller and sharper the needle, the less will be the injury to

the lung. With this idea in mind we have been using for many years a small sharp needle for the initial operation as well as for refills. For approximately 25 years, I personally performed nearly all initial operations in three different institutions using this method without any serious mishap. We use the same needle for injecting the anesthetic and the induction of pneumothorax. Usually 1¼ inch needle is long enough to reach the pleural space and the gage is 21, only slightly larger than the size used by others for anesthesia.

It is being argued that a dull needle will push the lung out of the way and for this purpose various needles have been devised and some operators use large bore needles with the point filed off. It is hard to believe that a needle that will penetrate the parietal pleura will spare the visceral pleura and in cases where the lung is adherent, obviously the lung cannot be pushed and it is bound to be punctured. It seems to me that the lung retracts somewhat by virtue of its own elasticity when air penetrates into the pleural space, and this will happen whether we use a sharp needle or a dull needle. At any rate, those who are afraid of a small sharp needle are using it anyhow when injecting the anesthetic, and that is practically the entire operation in our method.

As far as later complications are concerned, it goes without saying that if fluid develops and the pneumothorax is ineffective, it is best to abandon the pneumothorax, allow the lung to re-expand, and perform a thoracoplasty later. But in cases of effective pneumothorax, I do not believe it wise to abandon the pneumothorax merely because of the development of fluid, even if the fluid contains tubercle bacilli. In many instances the fluid will clear up after several aspirations and air replacements and it is possible to continue the pneumothorax.

Cases with frank empyema present an entirely different problem but here too, if the lung condition is not under control, the indications are to get the patient in good enough condition to stand a thoracoplasty. But in such cases where the pneumothorax has been effective and the only problem is the treatment of the empyema, the situation is different. If we are to believe various statistical reports, we would assume that empyema cases are entirely hopeless unless they are subjected to surgery. There are reports of 90 to 100 per cent mortality in non-operative cases. I am inclined to believe however, that the vast majority of patients died from progressive tuberculosis rather than from the empyema. Our own experience has not been that bad. In 1934, I reported a series of 21 cases of empyema treated conservatively, with 11 clinical recoveries. These patients were well at least five years after discharge from the sanatorium.

More recently, I reviewed 51 patients from the Los Angeles Sanatorium that were treated by me personally in a conservative manner. Of these 51 patients, 15 were working for at least two years. Eleven had their collapsed lung reexpanded and the empyema cavity closed. Three had a dry pneumothorax which was continued. Twenty were dead, but in only two or three cases could the empyema be considered the major cause of death. The remainder, were unimproved.

If these figures prove anything, it is that when considering surgery in a case of tuberculous empyema, it is a mistake to approach the problem with the idea that the patient is doomed if he does not submit to surgery. Whether or not our statistics would have been better had we employed surgery more frequently, it is impossible to state. But, given a patient with a large pneumothorax pocket, with a small amount of pus, whose lung condition is under control and who is doing well clinically, I found it difficult to advise the multiple surgical operations recommended for the obliteration of the pneumothorax. I doubt whether the patient would benefit much by converting a large pneumothorax space containing a small amount of pus to a small pneumothorax with pus and possibly a draining sinus.

Some statistics about patients who were subjected to surgery are exceptionally good, showing as much as 60 to 80 per cent cures, but obviously, these were selected risks because it is hard to find any group of cases without any complication in whom we can obtain such a large number of cures.

Heparin in Empyema
Report of a Case

ANGUS E. MacMILLAN, M.D., F.C.C.P.
Chehalis, Washington

In the treatment of pulmonary tuberculosis few complications are more perplexing than a mixed infection empyema. Very often aspiration is extremely difficult if not impossible because of the formation of a gelatinous substance within the cavity which often forms into long fibrinous strings or clots. These block the needle when aspiration is attempted. Occasionally one may see before the fluoroscope long strings of fibrin hanging from the parietal pleura and swinging in the fluid as the patient is flexed from side to side. In infections of the pleural cavity following pneumonia, cases are seen where even after a rib is resected and a tube inserted there is no drainage until the tube is removed and long strips of fibrin removed with a forceps. Howlett and Lester[1] recently reported two cases of Staphlococcus infection of the pleura in tuberculous patients successfully treated by penicillin. They state that in one case they were only able to aspirate once.

R. B., a girl twenty-one years of age, had been a patient in our sanatorium for eighteen months with a tuberculous involvement in the apex of the left lung. She had a satisfactory pulmonary collapse, although the base of the lung was adherent to the chest wall. In January 1946, she was allowed to attend business college in a neighboring city but returned for refills at intervals of two weeks. On March 14th, while typing with a heavy machine she experienced a sharp pain in her left side and a severe catch on breathing. She remained home in bed but her temperature arose to 103° and on March 18th she was returned to the sanatorium. X-ray films of her chest showed an increased collapse and some fluid forming at the bottom of the free pleural cavity. Penicillin was started with 10,000 units every three hours given parenteraly. Her temperature went to 104° and she had several severe epistaxes. By March 23 the fluid had greatly increased and she complained of pressure and shortness of breath. Aspiration was then decided upon. Only 20 cc. of a thick yellow fluid was withdrawn as the needle was continually blocked and had to be withdrawn several times during the aspiration of this small amount. This fluid clotted in a glass to the consistancy of jelly within a few minutes after its aspiration. A culture showed a pure staphylococcus infection. The following day aspiration was again tried with a much larger needle but with no better results.

On March 25, after aspirating 2 cc. of fluid, 2½ cc. of heparin in 10 cc. of saline solution was injected into the pleural cavity and allowed to remain for two hours. The patient was instructed to change her position

every fifteen minutes. At the end of this time 200 cc. of fluid was aspirated with no obstruction or difficulty of any kind and replaced by 10,000 units of penicillin. The fluid withdrawn did not clot or thicken when left standing in a glass receptable for several hours. Her blood clotting time (test tube method) was eight and one half minutes before injection of the heparin and the same when the heparin was withdrawn. From March 25th to May 25th the pleural cavity was aspirated daily, washed with normal saline, and 10,000 units of penicillin injected through the aspirating needle and allowed to remain. The intermuscular injections of 10,000 units every three hours started on March 19th, were also given until April 14th when they had to be discontinued because of the development of a severe urticaria accompanied by an arthritis in her right shoulder and elbow.

By May 25th the pleuritic fluid had decreased to such a small amount that aspiration became impossible. The urticaria and arthritis having disappeared by this date, the intermuscular injections of 10,000 units of penicillin every three hours were again given. Patient also received one capsule of benadryl orally each day. These were continued until June 15th without any reappearance of the urticaria or arthritis. At this date all treatment was discontinued. Three cultures of the aspirated fluid taken between May 20th and 25th had been negative to staphylococcus. Her temperature has been normal since May 15th and she has regained all weight lost during the first weeks of her secondary infection. Her lung is expanding satisfactorily and a very small pneumothorax remains containing not over 5 cc. of fluid. Patient has no sputum and the erythrocyte sedimentation rate is normal.

SUMMARY

Few conclusions can be drawn from one case but it does seem that heparin can safely be used in the pleural cavity without effecting the blood coagulation time of the patient. Guernar Bauer[2] in his splendid article "Heparin in Venous Thrombosis," reported on the use of very large doses of heparin intravenously without any ill effects. Lehman and Boys[3] injected 120 cc. of heparin in the abdomen following operations to prevent adhesions. Undoubtedly much larger amounts of heparin can safely be used in empyema cases than was used in the case just reported and allowed to remain longer before being withdrawn. It may also be to advantage to use it soon after the infection appears with a view of preventing adhesions which anchor the lung and retard or prevent its expansion.

RESUMEN

No es posible sacar muchas conclusiones basadas en un solo caso; pero parece que sí puede usarse sin peligro la heparina en la cavidad pleural sin afectar el tiempo de coagulación sanguínea del paciente. Guernar Bauer,[2] en su espléndido trabajo titulado "La Heparina en la Trombosis Venosa," informó sobre el uso de grandes dosis de heparina por la vía intravenosa sin mal efecto

alguno. Lehman y Boys[3] inyectaron 120 cc. de heparina en el abdomen después de operaciones, con el fin de evitar la formación de adherencias. Es indudable que en casos de empiema pueden usarse sin peligro cantidades mucho más grandes de heparina que la que se usó en el caso que se acaba de presentar, y se pueden dejar en la cavidad pleural por más tiempo antes de extraerlas. También puede ser conveniente usar la heparina poco después de aparecer la infección, a fin de evitar la formación de adherencias que sujetan el pulmón y demoran o impiden su expansión.

REFERENCES

1 Howlett, K. S., and Lester, D. E.: "Penicillin in the Treatment of Pyogenic Empyema Complicating Therapeutic Pneumothorax," *Am. Rev. Tuberc.*, 51:546, 1945.
2 Gunnar Bauer, Mariestad, Sweden: "Heparin in Venous Thrombosis," *J.A.M.A.*, May 18, 1946.
3 Lehman, Edwin P., and Boys, Floyd: "Clinical Use of Heparin in the Peritoneum for the Prevention of Adhesions: Report of Fourteen Cases," *Arch. Surg.*, 43:933-945, (Dec.) 1941.

Cor Pulmonale

G. F. STRONG, M.D.
Vancouver, B. C.

Twenty-five years ago cor pulmonale was usually taken to indicate the right-sided heart failure associated with emphysema or those forms of congenital heart disease in which the right ventricle bore the chief burden. Over the intervening quarter century there has developed a broadened concept of the term "cor pulmonale," so that it now means any type of heart disease associated with, or resulting from, obstruction, acute or chronic, in the pulmonary circulation. We now have *acute cor pulmonale* which most often results from sudden obstruction in the pulmonary circulation, either from embolism or thrombosis, and which may also be used to designate acute forms of right heart failure from any cause. There is in addition the much commoner *chronic cor pulmonale* which occurs in a number of cardiac and pulmonary disorders in which there is strain on the right ventricle. Frequent among these is mitral stenosis, in some cases of which the best examples of chronic right heart failure develop, as evidenced by increasing venous congestion, enlargement of the liver, and peripheral edema. Other examples of chronic cor pulmonale occur in those cases of congenital heart disease in which right ventricular failure dominates the clinical picture. This type of heart disease also is found in those rare instances of pulmonary arterial or arteriolar sclerosis, some of which are called Ayerza's disease. Further examples of chronic cor pulmonale are to be found in certain chronic lung conditions, especially those associated with fibrosis. This latter group includes emphysema, bronchiectasis, pneumoconiosis, especially silicosis, and pulmonary tuberculosis, in all of which interference with the pulmonary circulation may occur to a greater or lesser extent.

Our knowledge of the lesser circulation is limited and we must depend on animal experimentation to provide such information as we have. From studies on the dog it is assumed that the pressure in the pulmonary artery is about one-sixth that in the aorta; that means about 18 to 20 mm. of mercury. The output of the right ventricle is the same as the left ventricle, or 3 to 4.6 litres per minute, or in the neighborhood of 50 to 60 cc. per beat. The total capillary surface in the lungs is estimated at 140 square metres

*Presented at the Annual Meeting, Pacific Northwest Chapter, American College of Chest Physicians, Vancouver, B. C., October 4, 1946.

and the vessels show a high degree of distensibility. The capacity alters with changes in intrathoracic pressure and is of course very sensitive to change in the relation between the minute volume output of the right ventricle and the resistance on the left side of the circulation.

From a clinical standpoint estimation of right ventricular function depends on a correlation of symptoms, physical signs and laboratory tests. The symptoms are those of dyspnea, weakness, cough, hemoptysis; the signs are increased venous congestion with prominent or pulsating cervical veins, enlarged liver, and edema; and in chronic cases, cyanosis and clubbing of fingers and toes, and ascites. The laboratory tests may reveal a polycythemia, and x-ray films show right ventricular enlargement. In this connection it is important to remember that early dilatation or hypertrophy of the right ventricle may escape roentgenological detection, particularly if associated with emphysema with flattening of the diaphragm—the "drop" heart. The electrocardiogram usually shows right axis deviation. Measurement of venous pressure which is a simple bedside procedure, will usually show a considerable elevation over the normal figures of 40 to 80 mm. of water. Circulation time tests are also helpful, the right ventricular time being measured by the injection of 5 min. of ether in 1 cc. normal saline into an arm vein. The time taken for the ether to reach the lungs is normally 3 to 9 seconds, and in right ventricular failure this is much increased.

Some recent experimental work by Eaton, Czebrinski and Smith,[1] if confirmed, raises interesting speculations. These observers have found that as an immediate reaction to systemic arterial blood loss there is pulmonary hemorrhage and edema, which causes an increase in peripheral venous pressure as well as an increase of pressure within the pulmonary artery. This increased venous pressure is developed within 30 to 40 minutes of the acute blood loss and reaches its peak in 50 to 60 minutes. Thereafter there is a gradual decline to less than normal levels. They point out the obvious danger of the administration of intravenous fluids during this period of increased venous pressure, and suggest that venesection during this early period might on occasion be life-saving.

Before leaving this part of our discussion we cannot fail to comment on the impunity with which the thoracic surgeon can ligate a pulmonary artery, even the main right or left branch, and compare that procedure with the devastating effect produced by sudden occlusion of the same vessel by embolus or thrombus. This is but one of the many challenging propositions that await solution.

Of the acute forms of pulmonary artery involvement, embolism is probably the commonest, though spontaneous intravascular

thrombosis does occur. The most frequent source of the embolus is a thrombus in the right auricle or in a peripheral vein. Embolism may involve a small branch of one pulmonary artery and the symptoms produced may be mild and transitory, or a larger branch may be occluded with severe shock and more prolonged symptoms. Sudden blocking of the pulmonary artery itself is usually fatal. The findings in these various kinds of pulmonary embolism are of course quite different; the clinical picture is too well known to need detailed description here. There is the acute blocking of a small branch, characterized by sudden pain in the chest, cough, and hemoptysis which in its typical form can be diagnosed by the intern or nurse. What is less well known is that pulmonary embolism can occur without chest pain (which is really only an indication of pleural involvement), without cough, and in some cases without hemoptysis. It is the atypical cases which will only be discovered by a constant clinical awareness of this frequent pulmonary complication. In those cases in which a larger branch of the pulmonary artery is suddenly occluded *shock,* with rapid pulse, drop in blood pressure, extreme dyspnea, and cyanosis, may develop very suddenly. Some of these may even show anginal or substernal pain. Where the occlusion is high sudden death may occur. The electrocardiogram has proved of value for diagnosis in some of these cases. The cause of the shock, the cardiographic changes, and death in these cases, has been and still is the subject of a great deal of study. As pointed out by Murnaghan, McGuire and White,[2] pulmonary embolism and acute cor pulmonale are not synonymous. In other words, sudden occlusion of a branch of the pulmonary artery may occur without any evidence of right heart strain. They point out that acute cor pulmonale is more apt to occur in those instances of pulmonary embolism associated with shock. While it is true that in acute cor pulmonale the electrocardiographic changes may be characteristic, if we take all cases of pulmonary embolism, many will show no changes in the cardiogram at all, or only minor and nondiagnostic deviations from the normal. In acute cor pulmonale these observers have described the following changes in the tracings as suggestive: right axis deviation, deep ST, $S\text{-}T_1$ segment may be low, S-T take off low in Lead Two, T2 flat or inverted, Q3 moderately deep, T3 inverted, T4 diphasic or inverted. The limb lead changes are suggestive of posterior infarction. Others,[3,4] have stressed the importance of multiple precordial leads, especially those from the right side of the chest, the right pectoral lead. In these leads the T wave in acute cor pulmonale is inverted and remains inverted longer than in the usual precordial lead, IV.F. The whole subject of the heart in pulmonary embolism has been well discussed by Currens and

Barnes.[5] They presented 307 autopsied cases of pulmonary embolism, of which 70 per cent occurred in surgical, 20 per cent in medical, and 10 per cent in cardiac patients. They made a special microscopic study of thirty cases and found five that showed significant changes; four showed myocardial infarction without evidence of occlusion or thrombosis of the coronary arteries; one showed a recent coronary thrombosis. In another there was infarction resulting from prolonged shock in which there was definite anoxia, persistent cyanosis, and decreased coronary blood flow. In two others electrocardiographic changes had been demonstrated but the myocardium showed no changes.

The cause of death in pulmonary embolism is not known but various theories have been advanced, including a pulmono-coronary reflex in which coronary spasm, with or without cardiac infarction, might occur. Currens and Barnes[5] suggest that death is due to the onset of ventricular fibrillation and think that the angina that sometimes occurs after pulmonary embolism is due to coronary spasm. They stress the importance of the fall in arterial blood pressure with the increase in heart rate, the rise in pressure in the right auricle with resulting increase in pressure on the coronary sinus, and the decreased flow of blood to the muscle of the right ventricle. In the latter connection they point out that since over 90 per cent of the blood supply to the walls of the right ventricle is returned by way of the Thebesian veins, a rise in intraventricular pressure will impede the coronary circulation in the right ventricle.

In valvular heart disease cor pulmonale may occur if the chief strain is on the right ventricle. The commonest example of this sort of cor pulmonale is that found in some cases of mitral stenosis. In these patients there is usually dilatation of the left auricle with resulting increase in pressure in the pulmonary veins, which leads to obstruction of the lesser circulation with right ventricular hypertrophy. The symptoms result from diminishing vital capacity and peripheral venous congestion. There is early rise in venous pressure and circulation time, arm to lung, is greatly increased; later all the peripheral evidences of chronic passive congestion, hepatomegaly, edema of extremities, ascites, and cyanosis appear.

Of the causes of cor pulmonale that have their origin in the pulmonary arterial system itself, embolism and thrombosis produce acute symptoms, whereas pulmonary arteriolar sclerosis produces chronic cor pulmonale. These cases, some of which are so-called Ayerza's disease, and others which may result from congenital cardiac anomalies which cause an increased pulmonary blood pressure with resulting arterial and arteriolar sclerosis, are

not common. Ayerza's original description attributed the changes in the pulmonary arteries to syphilis. There are in addition other causes for pulmonary arteriolar sclerosis, among which must be included those conditions in which an increased pressure in the pulmonary artery is long maintained. These patients present a marked degree of cyanosis, are called "black cardiacs," and suffer an increasing disability from right heart failure; hemoptysis may also be common.

The cases of congenital heart disease that show cor pulmonale are those in which the abnormalities are such as to throw a constant and an increasing strain on the right heart. These usually fall in the Abbott group III,[6] the cyanosed group, and are clinically characterized by cyanosis, clubbed fingers and toes, increased venous pressure, and increased right ventricular (arm to lung) circulation time. Examples of this type of congenital heart disease are seen in cases of pulmonary stenosis, particularly those in which the narrowing is of considerable degree and is associated with a defect in the septum. The rarer cases of the Tetralogy of Fallot show a chronic cor pulmonale with right ventricular hypertrophy, resulting from pulmonary stenosis, defective intra-ventricular septum, and dextro-position of the aorta. Interest in pulmonary stenosis has increased because of the recent work of Blalock and Taussig,[7] suggesting a surgical method of relieving the cyanosis in these cases by the anastomosis of a branch of a systemic artery to the pulmonary artery.

There is another group of diseases which can produce a chronic form of cor pulmonale. These are the chronic diseases associated with more or less fibrosis of the pulmonary tissue, capable of causing obstruction to the blood flow through the lesser circulation. Included in this group of disorders are emphysema, bronchiectasis, pneumoconiosis, the commonest form of which in this area is silicosis, and pulmonary tuberculosis.

Emphysema, which may be the senile type or may be associated with bronchial asthma or chronic bronchial infection, has been the subject of much argument. The older clinicians coined the phrase "emphysema heart" and by this they meant a form of chronic cor pulmonale. On the other hand we all know that emphysema may exist for years with little evidence of heart strain of any sort; in fact, many of the most typical cases of chronic emphysema show small hearts. The x-ray evidence of right heart enlargement may be obscured by the emphysema itself with resulting flattening of the diaphragm, producing the so-called "drop heart" which may appear small in the film but actually show right ventricular hypertrophy. It is probably true that this impression is enhanced by the fact that when failure develops and enlarge-

ment of the right ventricle might be found, these patients fail rapidly and die from a non-recurrent form of heart failure in a short time. The studies of Parkinson[8] have shown that in emphysema the strain and resulting enlargement affect the outflow tract from apex of the right ventricle to pulmonary artery. Griggs and his associates[9] found evidence of right ventricular enlargement in 30 per cent of autopsies on all patients with emphysema and evidence of right ventricular enlargement in 28.9 per cent, and death from congestive failure in 22.3 per cent of patients dying with *only* pulmonary emphysema. It must be obvious therefore that there is an "emphysema heart" even though it is not a clinically common form of heart disease.

As to the role of other forms of chronic pulmonary disease in the production of chronic cor pulmonale, Griggs and his associates[9] found that chronic bronchitis alone was a negligible factor. On the other hand, where bronchiectasis occurred, they found that 16 per cent of all cases showed right ventricular hypertrophy, and where bronchiectasis was the only pulmonary disease, right ventricular enlargement occurred in 8.8 per cent, and the patient died from congestive heart failure in 4.4 per cent. This is added reason, in selected cases, for the early and adequate surgical treatment of bronchiectasis. In pneumoconiosis they found right ventricular hypertrophy in 43 per cent of all cases, and where the pneumoconiosis was the only pulmonary lesion they found right ventricular enlargement in 54 per cent, and death from congestive failure in 50 per cent.

Silicosis, which is a comparatively frequent clinical finding in this area because of the number of men engaged in hardrock mining, produces a widespread pulmonary fibrosis. These patients are susceptible to tuberculosis and this complication is common. Clinically, chronic cor pulmonale is not often found as a result of silicosis alone. Study of the chest x-rays of a large series of cases of silicosis by Vrooman[10] shows that cardiac enlargement is rare. It may be that some of these patients will ultimately die of right heart failure and show at autopsy right ventricular enlargement, but during life such right-sided heart involvement may not be apparent.

The relation of pulmonary tuberculosis to cor pulmonale is not clear. As in chronic emphysema, most patients with pulmonary tuberculosis, even those showing extensive pulmonary fibrosis, are more apt to show small rather than large hearts. It is of interest to note that Griggs[9] did find evidence of right ventricular enlargement in 3.75 per cent of cases of pulmonary tuberculosis, and thought that 1.8 per cent died of congestive heart failure resulting from chronic cor pulmonale. While these percentages are small,

the much greater incidence of tuberculosis in the general population means that this disease must be considered among the causes of cor pulmonale.

Diagnosis

The question of the diagnosis of cor pulmonale, in general, may be a more or less academic one. There is one form of acute cor pulmonale in which the differential diagnosis from coronary thrombosis may be important. As has been frequently pointed out, *awareness* of the ever present possibility of sudden pulmonary embolism is one of the most important factors in the differential diagnosis. There are instances in which because of sudden collapse, with tachycardia, fall in blood pressure, cyanosis, and respiratory distress, the picture of acute cor pulmonale from pulmonary embolism may closely simulate cardiac infarction. Anginal pain can also occur. The electrocardiographic findings in some of these instances may be the only means of differentiation. Fortunately treatment is not so dissimilar as to make early distinction between these two possibilities crucial. Oxygen will benefit both. Papaverine intravenously along with the early use of atropine is indicated in pulmonary embolism, and morphia in cardiac infarction. Both should benefit from aminophylline by vein.

Since cor pulmonale seems to be increasing and is certainly a more frequent diagnosis now than years ago, some thought should be given to those measures that may be adopted to prevent this disorder. It is obvious that we cannot prevent *all* the conditions that lead to cor pulmonale. In discussing the prevention of pulmonary embolism it is important to remember that this may develop in surgical or medical patients. In the former early ambulation, leg exercises, early ligation if evidence of peripheral venous thrombosis is discovered, are the important measures. The question of venography should receive more consideration since it may be the only method by which a silent peripheral venous thrombosis, phlebothrombosis, may be discovered. Actual surgical removal of the embolus from the pulmonary artery, while occasionally attempted, is not practical.

In medical patients the approach is somewhat different. In spite of all the recent papers decrying rest in bed, there are some medical conditions in which long periods in bed are necessary. Fortunately, not all patients confined to bed develop silent venous thrombosis from which pulmonary embolism may occur. There are instances, however, in which, with patients confined for periods of several weeks, peripheral venous stasis with thrombosis does occur. These patients may have an alteration in the coagualability of their blood, and it is for them that preventive meas-

ures should be considered. The routine use of the anti-coagulants, dicoumarin or heparin, in bed patients has its advocates, though the value of this method of treatment must await controlled studies. Where evidence of venous thrombosis has developed the anticoagulants or ligation, or both, may be indicated. Venography to establish the patency of the venous system, followed by regular and orderly leg exercises should certainly be of value in patients lying still in bed. In dealing with the chronic pulmonary disorders that produce cor pulmonale, prevention is in part to be found by earlier surgical treatment of the affected portions of the lung.

SUMMARY

There has been attempted a description of right heart failure called cor pulmonale which, while in the chronic forms, not always a clearly defined clinical entity, is much commoner than generally realized. Chronic cor pulmonale may present the clinical picture of general or "whole" congestive heart failure, although there are to be found examples of pure right ventricular failure. It is acute cor pulmonale which deserves most attention because in some cases it may be preventable. It may be confused with cardiac infarction and the differential diagnosis, including further study of the electrocardiographic changes, should receive more attention. The increase in the clinical and experimental investigation of venous thrombosis, both phlebothrombosis and thrombophlebitis, should add to our knowledge of methods of preventing pulmonary embolism. As our clinical awareness of this form of heart trouble increases, we will discover more frequent examples of acute cor pulmonale.

RESUMEN

Se ha intentado describir la insuficiencia cardíaca derecha, llamada cor pulmonale, cosa que, aunque no sea siempre clínicamente bien definida en sus formas crónicas, es mucho más común de lo que generalmente se supone. El cor pulmonale crónico puede presentar el cuadro clínico de insuficiencia cardíaca congestiva general o "entera," aunque existen ejemplos de insuficiencia del ventrículo derecho solo. El que merece más atención es el cor pulmonale agudo, porque en algunos casos puede ser evitable. Es posible confundirlo con el infarto cardíaco y, por lo tanto, debe recibir más atención el diagnóstico diferencial, incluyendo un estudio más detallado de las alteraciones electrocardiográficas. El acrecentamiento en la investigación clínica y experimental de la trombosis venosa, tanto flebotrombosis como tromboflebitis, se debe añadir a nuestro conocimiento de los métodos de evitar la embolia pulmonar. Con el aumento de nuestros conocimientos clínicos sobre esta forma de

cardiopatía, descubriremos con más frecuencia ejemplos de cor pulmonale agudo.

REFERENCES

1 Eaton, R. M., Czebrinski, E. W., and Smith, J. R.: "Observations on Pulmonary Arterial Pressure and Peripheral Venous Pressure Following Arterial Blood Loss," *J. Thor. Surg.*, 14:339, (October) 1945.

2 Murnaghan, D., McGuire, S., and White, P. D.: "Pulmonary Embolism with and without Acute Cor Pulmonale, with Special Reference to Electrocardiogram," *Am. Eeart J.*, 25:573, 1943.

3 Sokolow, M., Katz, L. N., and Muscovitz, A. N.: "Electrocardiogram in Pulmonary Embolism," *Am. Heart J.*, 19:166, 1940.

4 Wood, P.: "Pulmonary Embolism: Diagnosis by Chest Lead Electrocardiography," *Brit. Heart J.*, 3:21, 1941.

5 Currens, J., and Barnes, C. R.: "Heart in Pulmonary Embolism," *Arch. Int. Med.*, 71:325, 1943.

6 Abbott, M. E.: "Atlas of Congenital Cardiac Disease," *Am. Heart Assoc.*, 1936, p. 12-14.

7 Blalock, A., and Taussig, H. B.: "Surgical Treatment of Malformations of the Heart," *J.A.M.A.*, 128:189, (May 19) 1945.

8 Parkinson, J., and Hoyle, C.: "Heart in Emphysema," *Quart. J. Med.*, 6:59, 1937.

9 Griggs, D. E., Coggin, C. B., and Evans, N.: "Right Ventricular Hypertrophy and Congestive Failure in Chronic Pulmonary Disease," *Am. Heart J.*, 17:681, 1939.

10 Vrooman, C. H.: Referee in Silicosis to Workmen's Compensation Board of British Columbia. Personal communication.

A Technic for the Automatic Maintenance of Constant, Controlled Pneumothorax*

(A Preliminary Report)

I. ALFRED BRECKLER, M.D.**
EDWIN R. LEVINE, M.D., F.C.C.P.***
Chicago, Illinois

In the administration of artificial pneumothorax re-expansion of the lung may dictate very frequent refills. It has been shown by Pinner,[1] Matsuzawa,[2] and others that absorption of intrapleural air begins very shortly after the time of its introduction. When there is a sufficient amount of normal, elastic lung tissue, a large proportion of this air may be absorbed in the first 24 hours. Consequently, early in pneumothorax, in most bilateral pneumothorax cases, and in such cases where pathology does not involve large portions of the lung parenchyma, there is a very marked tendency to rapid re-expansion. This necessitates complete inactivity of the patient since muscular activity increases the rate of re-expansion and necessitates frequent needling of the chest.

In an institutionalized patient this is not a serious objection; however, there are places and there are circumstances when it is difficult to keep a patient at bed rest, and it is often difficult to give pneumothorax refills with the frequency required to maintain adequate collapse. For this reason it has long been the wish of chest men that an apparatus be designed that would introduce air as required to prevent rapid re-expansion of the lung and make repeated chest punctures unnecessary. Such an apparatus must maintain the intrapleural pressure desired, and the type of material introduced into the chest must not cause irritation, infection, or other undesirable complications.

The Pneumothorax Pressure Regulating Device

The machine, shown in Figure 1, is composed of magnesium alloy and weighs less than one pound. It has a diameter of 3½ inches and stands about 3 inches high.

Essentially, the apparatus is composed of two chambers, separated by a rubber diaphragm (Fig. 2). Chamber A is in direct

---

*From the Chest and Surgical Services, Michael Reese Hospital, Chicago, Illinois, and Winfield Hospital, Winfield, Illinois.
**Aided by a Fellowship in the Dazian Foundation for Medical Research, New York, New York.
***Director, Chest Service, Michael Reese Hospital and Medical Director, Winfield Hospital.

communication with air. Chamber B is airtight and communicates directly with the intrapleural space. The rubber diaphragm C separates the two chambers and is made airtight except where a needle valve (D) pierces the diaphragm. A seal is effected at this point by having the needle valve impinge on a seat of soft rubber (E). An airtight L tube (F) communicates with Chamber (B). The vertical arm fits the lumen of a thick-walled rubber tube which eventually is connected to the intrapleural catheter. The horizontal arm is covered with a snugly fitting rubber cap which may be removed so that a water manometer may be placed in series to read chest pressures. Impinging upon the diaphragm on the chamber B side is a spring of known strength (G). The pressure which the spring exerts on the diaphragm is controlled by turning a screw (H). This portion of the machine is rendered airtight by the addition of a snugly fitting sleeve of rubber (I).

It can be seen that by varying the pressure exerted by the spring (G), the summation of intrapleural pressure and spring pressure on the chamber B side of the diaphragm can be made to equal

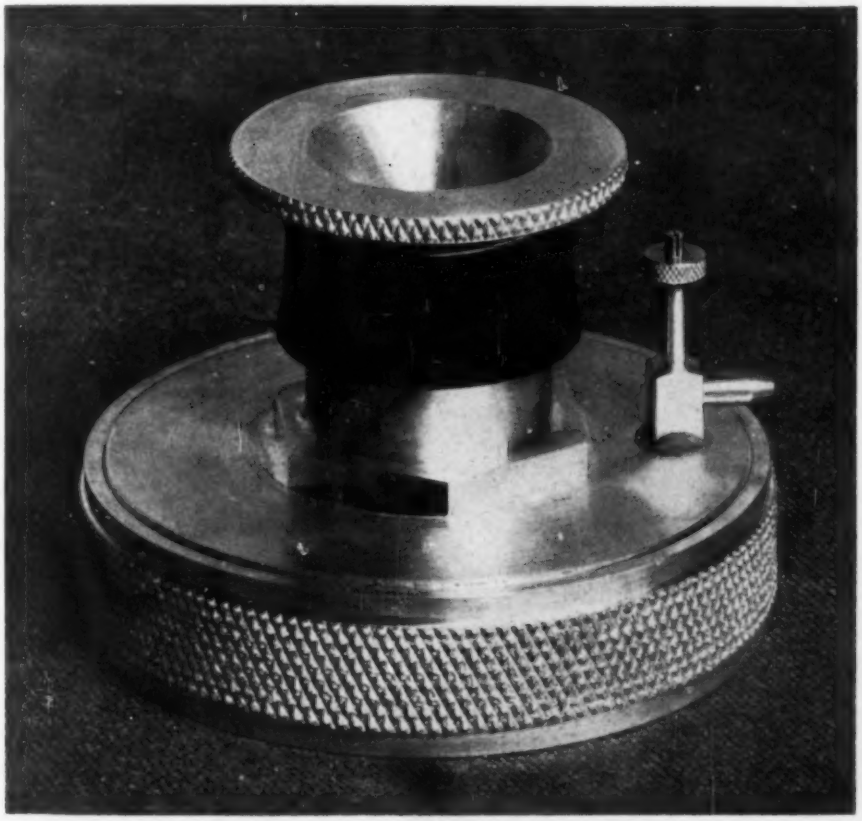


FIGURE 1: The constant pressure regulating device. The instrument is quite light, being made of magnesium alloy.

atmospheric pressure, which exerts its force on the chamber A side of the diaphragm. When such a balance ensues, the needle valve (D) is seated firmly in the rubber cushion (E), and the two chambers are completely separated. Chamber A communicates with air, and chamber B with the intrapleural space. When air is absorbed by the pleura, the intrapleural pressure decreases. Now the sum of forces pushing on the "B" side of the diaphragm *is less than* atmospheric pressure. The needle valve moves away from its seat, and air moves through it into chamber B and into the intrapleural space until the pressures are again equal on both sides. The valve then seats and closes.

It must be made clear that the machine is set to control an *inspiratory* pressure. During expiration the summation of forces in Chamber B will be greater than atmospheric pressure (Chamber A pressure). Thus, the valve will always stay closed until on inspiration the pressure exceeds the negative pressure adjusted for. At this point atmospheric pressure will force the needle valve away from the rubber cushion. Air will move into chamber B until the intrapleural pressure falls below the set value of negativity.

The spring tension, and thus the intrapleural pressure, may be set in the following fashion: A large syringe is attached to the vertical arm of the L tube (F) and the water manometer to the horizontal arm. Air is withdrawn from chamber (B) by the syringe. The difference of gas pressure between chambers A and B is made up by the spring pressure. Thus, when the water column in the

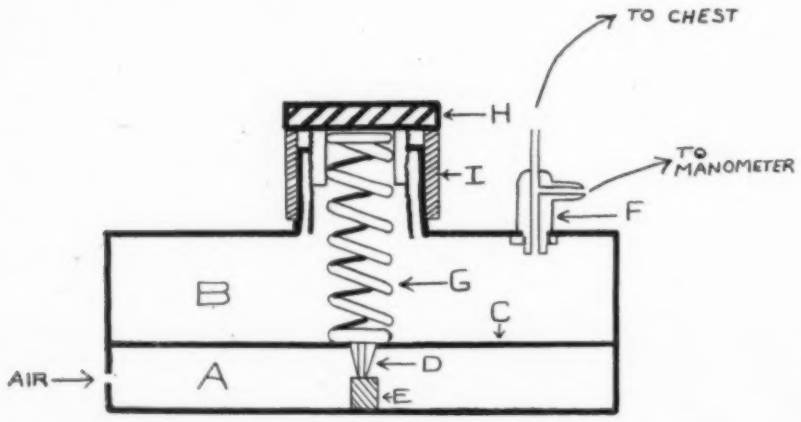


FIGURE 2: Cross section of the constant pressure regulating device.—(a) The lower chamber which communicates with air.—(b) The upper chamber which is airtight and communicates with the intrapleural space.—(c) The rubber diaphragm.—(d) A small needle valve which pierces the diaphragm and allows air to move from chamber A to chamber B.—(e) A small rubber cushion on which the needle valve sits.—(f) L tube, on arm of which leads into the chest, the other may be connected to a water manometer for pressure readings.—(g) Calibrated spring which rests on the diaphragm.—(h) Gnarled screw which when turned will increase or decrease spring tension.—(i) Rubber cuff.

manometer stabilizes at a negative pressure, a reading of the *difference* between intrapleural (B) and atmospheric (A) pressure is obtained. This is the precise pressure exerted by the spring, and may be read directly. So, if a *negative* intrapleural pressure of −10 cm. (i.e., 10 cm. of water less than atmospheric pressure), is desired, one need only to adjust the tension of the spring (G) by the screw (H) until a steady manometer pressure of −10 cm. is registered. It is possible to secure a range from 0 to −35 cm. of water pressure.

The Chest Catheter

An investigation soon revealed that rubber, metal, or woven catheters were far too irritating when left in situ in tissue. Of the available plastic materials studied, methacrylate and polyvinyl tubing were found unacceptable. Polyethylene seemed to answer the purpose quite well.

Polyethylene is a long-chained polymer of ethylene with a molecular weight of about 18,000. It was produced commercially in 1943.[3] The tubing used in our first two cases had an internal diameter of 22 gauge and an outside diameter of about 16 gauge. We then changed to a larger tubing which had an internal diameter of 17 gauge and an external diameter of 14 gauge.* It is a slightly cloudy, tough, flexible material, easily sterilized in 70 per cent alcohol solution. It cannot be heat sterilized. When samples of the tubing were left in situ in the thoracic cage of dogs for periods up to one month, there was no gross evidence of tissue irritation either along the catheter tract or in the pleural cavity. Microscopic studies were not done. The evidence of the low tissue irritation of polyethylene is supported by several other investigations. Meyer[4] used polyethylene for intravenous catheterization and reported minimal tissue response. Ingraham, Alexander and Matson[5] reviewed the use of plastics in surgery, and more recently conducted an exhaustive study of tissue reaction to polyethylene.[3] They feel this plastic is by far the least irritating to tissue. With this information it was felt sufficient evidence had been accumulated to introduce a polyethylene catheter into the intrapleural space of the human.

Technic

The technic does not differ markedly from that of pneumothorax. A 14 gauge needle which has been threaded through a flat rubber disc is inserted into the pleural space. Through the lumen of the needle the special polyethylene catheter is inserted

*Polyethylene tubing known as "aeroflex" was kindly furnished by the Anchor Plastic Corp., 533-541 Canal Street, New York, New York. The specific size of the larger tubing is .55" x .095".

until 5 cm. to 6 cm. of the catheter lies within the pleural space, and by pushing down gently on the catheter and withdrawing the needle, the catheter is left in the chest after the needle has been withdrawn.

The rubber disc through which the catheter is now passing is applied to the surface of the skin and affixed there by cellulose tape (Fig. 3). The catheter is fixed firmly in position, and a blunt 17 gauge needle is gently threaded into the lumen at the distal end of the catheter. A rubber tubing is attached to this needle by a luer lock, and the distal end slipped over the vertical arm of the L tube on the apparatus. The whole apparatus and tube may be placed in a pocket, pinned to an article of clothing, or hung on a belt. No special precautions need be taken.

CASE REPORTS

Case 1: E.S. A 19 year old white female. Pneumothorax was established on the right on October 7, 1946 and left pneumothorax induced on June 16, 1947. Because of the presence of lower lobe cavitation on the right, it was necessary to maintain adequate collapse not only in the right upper lobe but also in that portion of the right lower lobe that contained the cavity. This was found to be extremely difficult to do and required pneumothorax refills every 3 or 4 days. Since the patient was also receiving pneumothorax on the left once a week, this made a rather complicated schedule. This program had been in effect for the past 4 months without any noticeable improvement in the ability of the pneumothorax to maintain collapse of the right lung.

On September 2, 1947 a catheter was inserted into her chest and attached to the constant pressure apparatus. Figures 4a, b, c and d shows the maintenance of pneumothorax space during the 16 day period. As may be observed, the space actually increased in size in this period. It is to be noted that this patient did not remain on complete bed rest, and that previously her lung showed almost complete re-expansion of the lower lobe in 4 days. There was no reaction in the pleura, on the skin or in general. No elevation of temperature occurred nor were symptoms

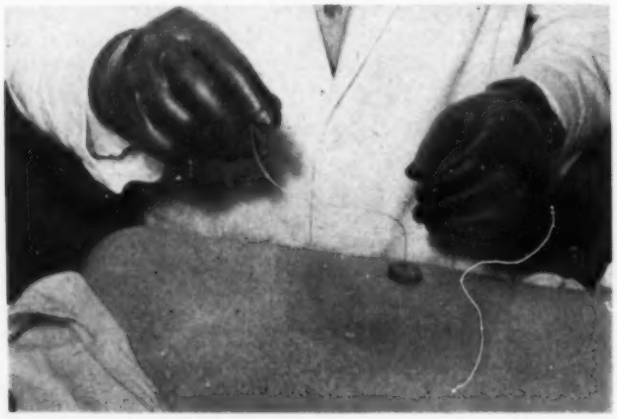


FIGURE 3: The polyethylene catheter may be seen running through the rubber disc and entering the chest wall.

noted by the patient. Pressure readings taken during this period showed a constant inspiratory pressure of −8, the level at which the apparatus was set.

Case 2: R.Mc. A 31 year old white female. Right-sided pneumothorax was established on August 5, 1946 and left pneumothorax on February 5, 1947. This case is almost a duplicate of Case 1 in pathology, indications, and complications. On September 2, 1947 a catheter was inserted

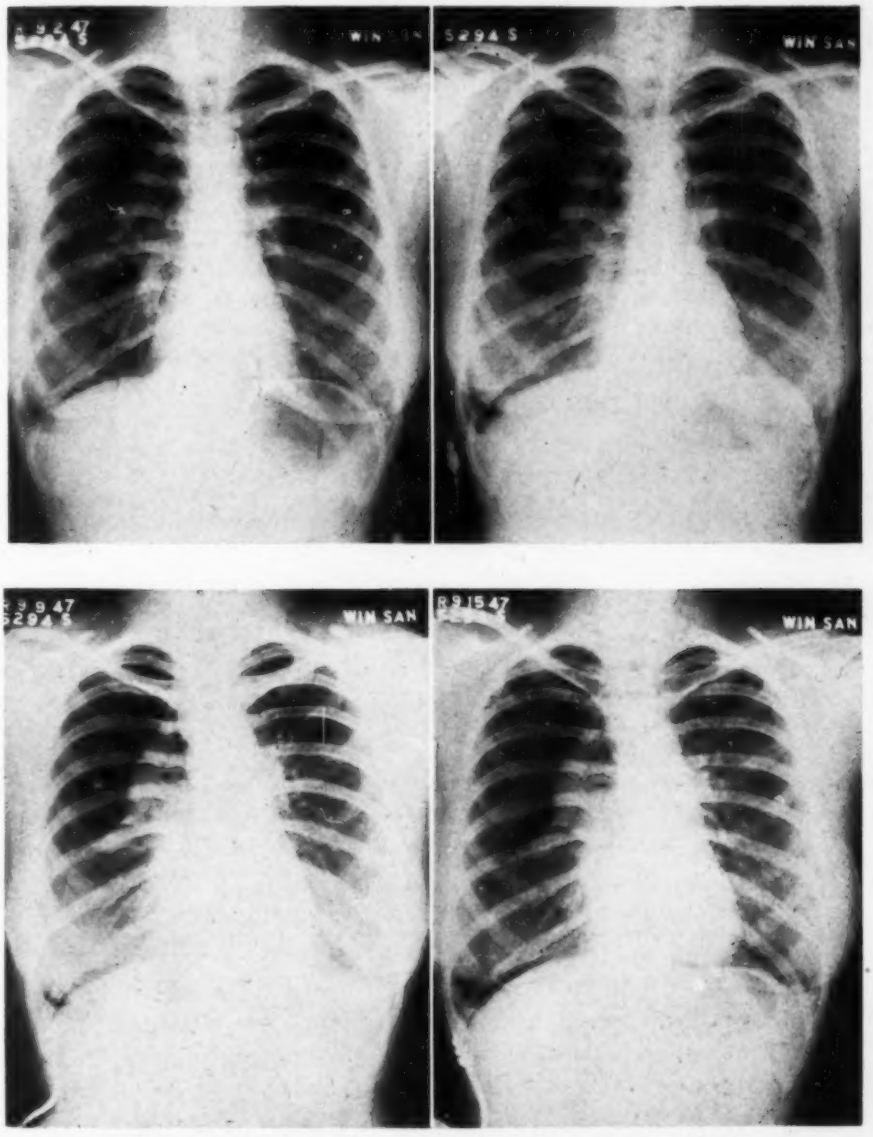


FIGURE 4: X-ray reproductions of films from Case 1, E.S. All of these films are taken in full inspiration.—(a) X-ray taken just prior to initiation of therapy.—(b) X-ray taken one day after therapy was begun. Note that the rubber tubing, luer-lok, and blunt needle may be seen.—(c) X-ray taken one full week after therapy was begun.—(d) X-ray taken two full weeks after therapy was begun.

into the right pleural space using the technic outlined above. For the first time since institution of pneumothorax there was an adequate collapse, maintained as in Case 1. There was no tendency to re-expansion although up to the day that the catheter was inserted, her lung re-expanded rapidly between refills. There were no symptoms, discomfort, no evidence of inflammation, or infection of the pleura or the chest wall, and no elevation of temperature.

Case 3: A.N. A 38 year old white male chosen because of a small lesion in the left midlung field with a small cavity. The rest of the lung was free of tuberculosis and represented the type of a case in which rapid re-expansion is expected following institution of artificial pneumothorax. Initial pneumothorax was done on September 29, 1947. Fluoroscopy on September 30, 1947 showed complete re-expansion of the lung. Refill (500 cc. of air) was given, and into the narrow space thus created a catheter was introduced and connected with the constant pressure apparatus. No further refills were given. Fluoroscopy 2 days later (October 2, 1947) showed approximately 50 per cent collapse of the left lung. Analysis of the pleural gas was done at that time. On October 3, 1947 there was some increase in the amount of collapse, and another gas analysis was taken with no introduction of air. These gas analyses, as shown in Table I, indicate a high oxygen concentration and a low concentration of carbon dioxide. At the end of one week the collapse was completely satisfactory in all respects. There were no symptoms up to this time.

TABLE I

Oxygen-Carbon Dioxide Analyses of Intrapleural Gas (Case 3)

(Figures expressed in terms of per cent of total volume)

	Oxygen	Carbon Dioxide
Second Day of Collapse	19.1	2.9
Fifth Day of Collapse	19.4	.71
Normal Volumes of Intrapleural Gases (Literature)		
D. Matzusawa	4.16- 1.97	5.82-6.3
M. Pinner	7.0 - 4.0	7.0
Coryllos, et al	6.0 - 2.1	6.0 -8.0

Oxygen-Carbon Dioxide values of intrapleural gases as determined from samples taken from Case 3, A.N. Previous determinations done on intrapleural gas of cases treated by the standard technic are included for comparison.

Discussion

The experience in these three cases indicates the following:

1. The use of a polyethylene catheter in the chest is not conducive to inflammatory reaction or infection, and is well tolerated by the patient.

2. Furthermore, it appears that the collapse of the lung may be

maintained without the necessity of pneumothorax refills as has been indicated by failure of the lungs of these patients to re-expand. That this is not an uncontrolled introduction of air is indicated by Figure 5, showing the constancy of intrapleural pressure when the apparatus is attached. It may be noted by the question marks on the chart that there were days when it was impossible to determine the pressure since no reading was obtained through the catheter.

Originally, we were quite disturbed by this, feeling that no air could be introduced. However, the maintenance of collapse and the further evidence of the gas content seemed to indicate that this inability to obtain pressure readings does not necessarily indicate a completely blocked airway. It appears that a small amount of liquid in the tube may interfere with small fluctuations of the air column. For this reason in the third case a somewhat larger tube was used, and no difficulty in obtaining pressure readings was encountered. It might be noted that there appeared to be no variations in the pressure, and that once the apparatus was set up a particular pressure could be maintained in the chest. Another evidence of ingress of air is found in the gas analysis on the third patient. It has been shown by several authors[1,2,6] that the average gas content in a pneumothorax space will range from 6 to 2.8 per cent oxygen and 6 to 9 per cent carbon dioxide, depending upon

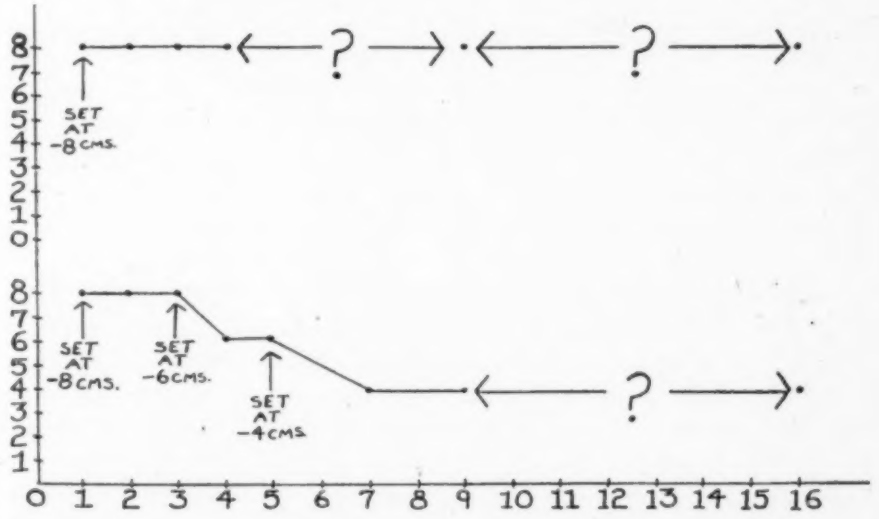


FIGURE 5: Intrapleural chest pressures. The upper graph represents data from Case 1, E.S. The instrument was set to maintain an inspiratory pressure of —8 cm., and it will be noted that whenever pressures were obtained, they were always —8 cm. The lower graph represents pressure readings from Case 2, R.Mc. The pressure was set at —8 cm. and stayed at this value for 3 days. When the pressure held by the machine was reduced to —6 cm. on the 3rd day, intrapleural pressures promptly fell. On the 5th day the intrapleural pressure promptly fell to —4 cm. and stayed there when the instrument was set at —4 cm.

the duration of collapse. It is felt that any oxygen percentage in a recent pneumothorax over 6 per cent is indicative of ingress of air. An oxygen percentage maintained in the vicinity of 19 per cent with a carbon dioxide below 3 per cent must be considered definite evidence that air is being constantly introduced into the chest.

It would seem likely that this technic would be extremely valuable in areas where it is difficult for patients to receive frequent refills as well as sections of the world where adequate beds for the number of pneumothorax cases are not present. It remains to be seen whether this will prove to be an advantage in every case of pneumothorax. One thing appears definite—that air can be introduced into the pleural space as it is needed, and the lung can be maintained in a state of constant collapse rather than a state of constant re-expansion and subsequent collapse. On the basis of any theory of efficacy of pneumothorax, a constant collapse is more desirable than an intermittently expanding lung.

DISCUSION

La experiencia en estos tres casos indica lo siguiente:

1) El uso de un catéter de polietileno en el tórax no produce reacción inflamatoria ni infección, y es bien tolerado por el paciente.

2) Además, parece que el colapso del pulmón puede ser mantenido sin necesidad de re-insuflaciones del neumotórax a juzgar por el fallo de los pulmones de estos pacientes a re-expanderse. Que no se trata de una incontrolada introducción de aire es indicado en la figura 5, que muestra la constancia de la presión intrapleural cuando el aparato está conectado. Se puede notar por el signo de interrogación en el cuadro, que hubo días en que fué imposible determinar la presión puesto que no se obtuvo al través del catéter.

Al principio, esto nos preocupó grandemente, pensando que no se podría introducir aire. Sin embargo, el mantenimiento del colapso y la subsiguiente evidencia del contenido gaseoso parecieron indicar que la imposibilidad de obtener registros de la presión no indicaba necesariamente que hubiese un bloqueo completo. Parece que una pequeña cantidad de líquido en el tubo puede interferir con ligeras fluctuaciones de la columna gaseosa. Por esta razón, en el tercer caso se usó un tubo algo mayor, y no hubo dificultad en la obtención del registro de las presiones. Se puede notar que no pareció haber variaciones en la presión, y que una vez que el aparato fuese instalado una presión determinada podría ser mantenida en el tórax. Otra evidencia de entrada de aire se encontró en el análisis del gas del tercer paciente. Ha sido demostrado por varios autores[1,2,6] que el gas promedio contenido en un neumotórax

varía entre 6 y 2 por ciento de oxígeno, y entre 6 y 9 por ciento de anhidrido carbónico, dependiendo de la duración del colapso. Se considera que cuando el contenido de oxígeno en un neumotórax reciente es más del 6 por ciento, indica entrada de aire. Un contenido de oxígeno mantenido en los alrededores del 19 por ciento, con anhidrido carbónico inferior a 3 por ciento, debe ser considerado como evidencia definitiva de que el aire está siendo introducido constantemente en el tórax.

Parece posible que esta técnica de extraordinario valor en lugares donde es difícil para los pacientes el obtener frecuentes reinsuflaciones, así como en partes del mundo donde no hay suficientes camas para los casos de neumotórax. Queda por demostrar si será ventajosa también en todos los casos de neumotórax. Una cosa parece definitiva—que el aire puede ser introducido en la cavidad pleural según es necesario, y que el pulmón puede ser mantenido en un estado de colapso constante, en vez de re-expansión constante y subsecuente colapso. Basándose en cualquiera de las teorías sobre la eficacia del neumotórax, un colapso constante es más deseable que uno intermitente.

REFERENCES

1 Pinner, Max: "Collapse Therapy," in *Medical Physics*, Year Book Publishers, Inc., Chicago, Ill., 1944.
2 Matsuzawa, D.: "Behavior of Pneumothorax Gases," *Quarterly Bull. Sea View Hosp.*, 2:363, 1937.
3 Ingraham, F. D., Alexander, E. and Matson, D. D.: "Polyethylene, a New Synthetic Plastic for use in Surgery," *J.A.M.A.*, 135-82, 1947.
4 Meyers, L.: "Intravenous Catheterization," *Am. J. Nursing*, 45:930, 1945.
5 Ingraham, F. D., Alexander, E. Jr. and Matson, D. D.: "Synthetic Plastic Materials in Surgery," *New England J. Med.*, 236:362-368, 402-407, 1947.
6 Coryllos, P. N., Konterwitz, H. and Levine, E. R.: "Clinical Application of Gas Analysis in Artificial Pneumothorax," *Am. Rev. Tuberc.*, 26: 153-161, 1932.

Dihydrostreptomycin in Experimental Tuberculosis*

(Preliminary Report)

B. L. FREEDLANDER, M.D., F.C.C.P., and F. A. FRENCH**
San Francisco, California

Dihydrostreptomycin is the reduced form of streptomycin, wherein the aldehyde group of streptobiosamine is reduced to a primary alcohol group. Dihydrostreptomycin is more stable than streptomycin and, in general, shows the same bacteriostatic action as streptomycin on most organisms.[1] Recently, Youman[2] reported dihydrostreptomycin as having the same tuberculostatic action in-vitro as streptomycin on strain H37Rv. In the following experiments dihydrostreptomycin hydrochloride and streptomycin hydrochloride were tested simultaneously for therapeutic effects in-vivo.

Method

Eight to twelve guinea pigs, averaging 380 gms. wt., were used to evaluate each compound. The animals were inoculated in the left groin with 0.01 mg. of H37Rv tubercle bacilli. The drugs were given in dosages of 10 mg.Δ twice daily subcutaneously to each animal. Smith[3] has shown the effectiveness of streptomycin when given in large dosages twice daily. The experiment was terminated at the end of 6 weeks. Pathological ratings were based on a maximum of 4 for each organ, with a maximum of 16 for all organs. Hemoglobin and weight determinations were made at the end of the experiment.

Results and Discussion

The results are summarized in Table 1. Dihydrostreptomycin is fully as effective as streptomycin under the conditions of this experiment. While the results are not conclusive there is a suggestion that dihydrostreptomycin may be slightly more effective than streptomycin in guinea pig tuberculosis. Bailey and Cavallito[4] have suggested that dihydrostreptomycin possesses no anti-bac-

---

*The present report is part of a cooperative investigation on tuberculosis, and has been supported by funds provided by the Committee on Medical Research of the National Tuberculosis Association, the California Tuberculosis Association, the San Francisco Tuberculosis Association and the Columbia Foundation of San Francisco.

**From the Harold Brunn Research Institute, Mt. Zion Hospital, San Francisco, California.

ΔThis is equivalent to approximately 50 mg./Kg. daily.

TABLE 1

Summary of in-Vivo Experimental Data

	AVERAGE							PATH. RATIO
			PATHOLOGY					
	Wt. Gain	HGLB.	Lymph Glands	Spleen	Liver	Lungs	Total Path.	CONTROLS / TREATED
Dihydro streptomycin	145	12.6	0.67	0.17	0.	0.42	1.25	5.5
Streptomycin	178	12.5	0.75	0.25	0.13	0.63	1.73	3.9
Controls	183	13.1	2.4	2.1	0.5	1.8	6.82	1.0

terial action per se, but is oxidized to streptomycin by the bacteria.

As factors of penetration and concentration maintenance in certain tissues may be more favorable with dihydrostreptomycin than with streptomycin, broader studies are indicated. In addition, other streptomycin derivatives, capable of in-vivo conversion to streptomycin may be of interest. Experiments are now in progress to determine blood concentration versus time curves of dihydrostreptomycin in comparison with streptomycin. There are many factors which determine the ability or inability of a drug to penetrate certain tissues. These factors include permeability behavior dependent upon molecular shape and size, the distribution of polar and ionic groups in the molecule, solubility, distribution coefficient, and the past history of the cells involved.

The brain is a unique organ in its behavior relative to the absorption of various nutrients and other agents across the blood-brain barrier.[7] There is no known rationale, at present, to the remarkable selectivity displayed. Streptomycin is not absorbed in appreciable quantities and has little or no clinical effect on lesions in the brain but suitable derivatives might be absorbed. Such agents, if active chemotherapeutically, might have a definite value in tuberculosis involving the brain, which organ is frequently involved in tuberculous meningitis.

The limitations of the clinical usefulness of streptomycin are due to the drug's apparent inability to penetrate all tissues in sufficient concentration. Corper[5] has shown that the concentration of streptomycin required to retard the growth of tubercle bacilli in-vitro in good nutrient medium can be attained in-vivo for only a brief period. He also questions whether streptomycin enters important organs in appreciable amounts for any significant time. The authors[6] have pointed out that streptomycin seems to be less effective in those types of tuberculosis, wherein caseation predominates. Caseous tissue might constitute a barrier to the

penetration of streptomycin. It is possible that dihydrostreptomycin might penetrate this caseous barrier in greater concentration. Animal and clinical experiments are contemplated to determine these distribution and local therapeutic factors.

SUMMARY

1) Dihydrostreptomycin was found as effective as streptomycin in the therapy of tuberculosis in guinea pigs.

2) The possible clinical significance of these results relative to overcoming the limitations of streptomycin is discussed.

REFERENCES

1 Bartz, Q. R., Controulis, J., Crooks, H. M. and Rebstock, M. C.: "Dihydrostreptomycin," *J. Am. Chem. Soc.*, 68:2163, 1946.
2 Youmans, G. P., cited in Reference No. 1, page 2166.
3 Smith, M. I., McClosky, W. T., Jackson, E. L. and Bauer, H.: "Chemotherapeutic Action of Streptomycin on Tuberculosis," *Proc. Soc. Expt. Biol. & Med.*, 64:261, 1947.
4 Bailey, J. H. and Cavallito, C. J.: "Mode of Action of Dihydrostreptomycin," *J. of Bact.*, 547, 1947.
5 Corper, H. J. and Cohn, M. L.: "Tubercle Bacillus and Fundamental Chemotherapeutic and Antibiotic Action," *Yale J. of Biol. & Med.*, 19: 1, 1946.
6 Freedlander, B. L. and French, F. A.: "Constitution of Chemotherapeutic Agents in Experimental Tuberculosis," *Am. Rev. of Tuberc.*, in press.
7 Drinker, C. K., Folch, J., Cobb, S., Gasser, H. J., Penfield, W. and Strecker, E. A.: "Psychiatric Research," *Harvard University Press*, Cambridge, Mass., 1947.

Kendall Emerson Retires

Blackstone Studios, Inc., New York, N. Y.

Aboard the special National Tuberculosis Association train to the annual meeting in Portland, Oregon, in June, 1928, was a handsome, dignified man who was somewhat of a newcomer among the workers in the Association. The famous Managing Director, Dr. Linsly R. Williams, introduced him as Dr. Kendall Emerson. During the next few days the rumor was extant that this gentleman was to succeed Dr. Williams. On October first of that year Dr. Williams resigned, and Dr. Emerson was elected to the managing directorship of the National Tuberculosis Association. With this announcement tuberculosis workers everywhere began to inquire about Dr. Emerson's past work. They did not question his ability or qualifications for the new position because they had confidence in those who had chosen him. Still they were anxious to know and tell others who he was. They found he was born in Northhampton, Massachusetts on June 27, 1875, and received the degree of Bachelor of Arts from Amherst College in 1897 and Doctor of Medicine from Harvard in 1901. After completing an internship in the Massachusetts General Hospital he began to practice orthopedics and general surgery at Worcester, Massachusetts. Later he limited his work to orthopedic surgery which he practiced until 1928. In those days there was still a great deal of tuberculosis of the bones and joints in this country and, therefore, Dr. Emerson's interest in tuberculosis must have been large and continuous since graduation from medical school. Doubtless he had seen many tuberculous cripples who had not been diagnosed in time that treatment could be of any avail. On the other hand, he saw many sufficiently early that he treated them successfully. During these years Dr. Emerson took an active part in the organized effort against tuberculosis in Massachusetts. Obviously he visualized a great opportunity in the whole field of tuberculosis with reference to early diagnosis, proper treatment and prevention and, therefore, the position offered by the National Tuberculosis Association appealed to him.

This Association was organized in 1904. Many persons had contributed mightily to its development, and it was a powerful health organization when Dr. Emerson assumed its management. If anyone had doubt as to the future of the Association under his direction it was promptly dispelled. His poise and equanimity, combined with a vast store of information, kindness to all, but firmness when occasion demanded, immediately gained for him the confidence of tuberculosis workers everywhere. Under his management the Association continued to develop rapidly. All of its worthwhile activities flourished. One project was scarcely finished when another was contemplated and activated. Dr. Emerson

soon was in demand among other nations of the world. He was called to various countries to attend conferences and give advice. He was probably responsible in no small way for 33 nations now having national tuberculosis associations. His ability to select people for key positions with the National Tuberculosis Association is reflected in the fine work accomplished by the various heads of departments and their co-workers. In 1928 the gross income from the Christmas Seal sales was $5,465,738.71, but in 1946 it was $17,075,608.44. This phenomenal growth in income has made greater and greater accomplishments possible. In 1928 the mortality rate from tuberculosis in the United States was 79, but in 1946 it was approximately 36 per 100,000. Obviously there has been a corresponding decrease in morbidity and infection attack rate.

Being a member of the Board of Directors of the National Tuberculosis Association when Dr. Emerson became managing director and serving in that capacity for twelve years under his management, and also serving for one year as president of the National Association and on numerous committees during his regime, I had an opportunity to observe him and his work at close range. Never was there a single occurrence to cause my confidence in and admiration for him to waver. It was obvious that he was handling a tremendous task most admirably. On occasions he was criticized for over-conservatism, but I never detected this quality in him to a fault. He was conservative but only to a degree consistent with the responsibilities of his position in guarding the welfare of the Association.

Not one of my numerous requests for information and advice was ever denied. Each one was honored in a most cheerful manner. As editor of a medical journal for the past fifteen years, I have asked Dr. Emerson to prepare an introduction for each special annual number on tuberculosis. His manuscript always arrived well in advance of the deadline, and not once did it fail to contain an important message to our readers. All of this is only voicing the admiration and respect which large numbers of physicians, nurses, public health workers, and laymen hold for Kendall Emerson.

For nearly twenty years he has contributed nobly and magnificently to the cause of tuberculosis control everywhere. Although he expressed a desire to retire some time ago, his loyalty demanded that he remain until a successor was appointed. After the final roundup is over and the history of tuberculosis is written, the name, Kendall Emerson, will loom large, and his contributions will receive their greatly deserved credit for the accomplishment.

J. A. M.

James E. Perkins Accepts

With the inevitable age retirement of Kendall Emerson a responsibility of great magnitude devolved upon those made responsible for the selection of his successor as managing director of the National Tuberculosis Association. These thoroughly dependable persons combed the country for the qualified person who might be available for this important position. When the committee announced that it had selected Dr. James E. Perkins and he had accepted the managing directorship of the National Tuberculosis Association effective January 1, 1948, his many friends were delighted and expressed their assurance of the continued success of the National Association. Because he has worked mostly with public health organizations and departments, he is not known widely among the tuberculosis workers of this country. Many of them are asking who he is. They are certain of his qualifications; otherwise he would not have been selected, but they want to become acquainted with their new leader.

James E. Perkins was born in Saint Paul, Minnesota on July 17, 1905. He attended the schools of that city and then entered the University of Minnesota, where he received the degree of Doctor of Medicine in 1930. While in school he developed a keen interest in public health, and upon acquiring his medical degree became epidemiologist in the Minnesota State Department of Health. He was offered fellowships to the Johns Hopkins School of Hygiene and Public Health in Baltimore, where he received the degrees of Master of Public Health in 1931 and Doctor of Public Health in 1933. In 1934 he was appointed epidemiologist with the New York State Health Department, and from 1935 to 1938 he served as District State Health Officer. For the next seven years Dr. Perkins was Director of the Division of Communicable Diseases. Then he was made Deputy Commissioner of Health of the State of New York in 1946, which position he held at the time of appointment to the National Tuberculosis Association office.

Throughout these years Dr. Perkins' interest in tuberculosis has been deep and abiding. It would almost appear that he has devoted his life to preparation for this new position. Indeed, the thesis which he prepared for the Public Health degree at Johns Hopkins School of Hygiene and Public Health under the late Dr. Wade Hampton Frost was entitled, "The Study of Tuberculosis Deaths in the Eastern Health District of Baltimore, Maryland, with Special Attention to the Economic Aspects." Another investigation report was entitled, "A Study of Tuberculosis Among Mexican Sugar Beet Workers in the Red River Valley of Minnesota." In 1936 he made a special study of tuberculosis deaths in Fulton

and Montgomery Counties in the State of New York, and in 1942 supervised a study conducted by Dr. Thomas D. Dublin on, "The Epidemiology of Tuberculosis in Columbia County, New York." Thus, he has been striking at the very heart of tuberculosis.

In future tuberculosis work there is probably nothing as important as its epidemiology. This is one of Dr. Perkins' chief interests. He is a member of the American Epidemiological Society and a Life Member of the American Public Health Association, in which he has served as Secretary of the Epidemiology Section from 1942 to 1946, and Chairman in 1947. Health education has always been an important activity of the National Tuberculosis Association. In this respect Dr. Perkins again qualifies admirably. He was associate professor of Public Health and Preventive Medicine at the Albany Medical College from 1937 to 1946, and Associate Editor of the American Journal of Public Health from 1942 to 1947.

How fortunate the National Tuberculosis Association is in that, at the proper moment, a man with such excellent training, with so much practical experience, with such a keen interest in tuberculosis, with such a delectable personality, with such a splendid record as an educator, and with such a long expectancy of service, should appear on the scene. Certainly everyone will rejoice in Dr. Perkins' appointment and support him undauntedly.

A preponderance of the members of the American College of Chest Physicians in this country are also members of the National Tuberculosis Association. The others will profit by becoming members and also contribute to a great cause. Moreover, members of the College of numerous other nations should take an active part in their National Tuberculosis Associations. Tuberculosis is one of the many chest diseases which the College has pledged itself to destroy.

J. A. M.

REPORT OF THE COMMITTEE ON THE MANAGEMENT AND TREATMENT OF DISEASES OF THE CHEST*

For some time this Committee has been occupied with the difficulty of evaluating therapy because of the known inadequacy of the present methods of terminology and because it was impossible to get any large group of men in the field to agree upon what was meant by any of the present diagnostic and therapeutic criteria. During this past year an attempt was made to create some new system of terminology as applied to both diagnostic and therapeutic criteria.

Tuberculosis was used as the disease in question. There was extensive discussion among committee members in the attempt to determine what factors existed in the patient with pulmonary tuberculosis that might in any way influence the necessity for therapy and the efficacy of therapy. We were attempting to find items that could be used in judging diagnostic or therapeutic procedures.

We found that we could not agree even in general on this problem which is admittedly much more simple than the over-all one. In our conversations with other men of great experience in the field, we found further sources of disagreement. As a result of this a list was made of all the various factors which were considered to play a major part in the course or treatment of pulmonary tuberculosis. These amounted to approximately 33 separate factors which were organized and made up into a questionnaire. A sheet of instructions was enclosed with these questionnaires explaining in sufficient detail what was meant by each one of the various terms used. The questionnaire follows:

Patients No. ..... Age ..... Sex ..... Color .....

Febrile ..... Non-febrile .....

(check appropriate space)

A. *Diagnosis N.T.A.*

1. Minimal ..... Moderately Advanced ..... Far Advanced .....
2. Status: 1 ..... 2 ..... 3 ..... 4 .....

B. *Diagnosis* (committee plan):

1. With Cavity
   a. Solitary cavity ..... b. Cavity with exudation .....
   c. Cavity with fibrosis .....
2. Without Cavity
   a. Simple exudation ..... b. Fibrosis ..... c. Miliary Foci .....
   d. Caseation
      1. Encapsulated caseous foci .....
      2. Caseous pneumonia .....
3. Extent and Location (circle to indicate involvement in area):

   R1 R2 R3

   L1 L2 L3
4. Activity:
   a. Active ..... b. Resolving ..... c. Stable .....

C. *Treatment* .....

.....

D. *Result* .....

.....

*Presented at the Annual Meeting, Board of Regents, American College of Chest Physicians, Atlantic City, New Jersey, June 5, 1947.

This was then sent out to sanatoria and tuberculosis services in various sections of this country and to Mexico with the request that each of these institutions fill out the forms for each of the 100 patients who were admitted beginning January 1945. Approximately 1,000 of these forms were returned.

The forms were analyzed to determine 1) whether all of these factors were equally important; 2) whether some factors appeared to play a more important part than others in our problem; and 3) to review once more the correlation between indications, treatment, and clinical results and the customary N.T.A. classification. This made it necessary to check each item against every one of the others. It soon developed into a tremendous mass of statistics. With what was considered proper sampling, we made a complete analysis and found that there was a consistent relationship shown by only a few of the many factors on the form. These were:

1) Temperature: Patients with elevated temperature, rectal, 101° F. or higher continued for a period of over one month. This group seemed to be associated with a bad prognosis regardless of any other findings. There was also a high correlation between this factor and the difference between bed rest and other therapy.
2) Cavity with exudation: In this pathological group there was a very marked difference between results with collapse therapy and results with bed rest.

TABLE I

	Treated	Non-Treated	Good	Unimproved	Dead
Febrile	34%	76%	40%	22 %	38 %
Non-febrile	41%	59%	77%	22 %	2 %
Cavity with Exudation	70%	30%	66%	22.5%	11.5%
Cavity with Fibrosis	25%	75%	75%	12.5%	12.5%
Non-cavitary	0	100%	100%	0	0

It was very interesting to note that some of the characteristics or factors that we were certain would be an important element in the efficacy of treatment, such as color, age, and so forth, do not seem to function *per se* but in relation to the above two factors.

After considerable discussion it seemed that a new terminology that might be of greater value would in addition to everything else have to include under separate categories the above two criteria. It was felt that for the purpose of evaluation of therapy and not, in any sense, an attempt to classify from the standpoint of pathogenesis or course that any pulmonary tuberculosis could be broken down into 3 groups.

These would be 1) non-cavitary; 2) cavity with associated fibrosis; 3) cavity with associated exudation. In each of these groups we would have to separate the febrile and non-febrile patients according to our explanation in group 1.

It is quite obvious that the extent of the disease would also play a very important part in individual cases, that in this preliminary survey

at least, there was not a marked difference until we reached cases with very extensive pathology. It is planned to make up forms embodying these principles and to attempt to thus classify a fairly large group of cases. Since this would be a pilot demonstration, we expect to use for purposes of differentiation only those methods of treatment as are generally accepted and where a favorable end-result is a matter of reasonably general understanding.

This will be compared with bed rest as the base line rather than a complete lack of treatment since we feel that this will give a more accurate measurement. The various sub-committees will do similar studies on the particular subjects under their jurisdiction. This will enable us to secure a mass of very valuable material. Whether it will be possible to evaluate any of the therapeutic measures or whether we simply end up evaluating our methods of criteria remains to be seen.

If our pilot demonstration yields any reasonable degree of accuracy, we will have material upon which we can make a statement of the relationships of indications, efficacy, and end-results of some of the more commonly used methods of treatment. It is hoped further that this year we will complete our first problem and present to the College for its approval a complete system of terminology, diagnosis, and treatment of tuberculosis.

Edwin R. Levine, M.D., Chicago, Illinois, Chairman
Donato G. Alarcon, M.D., Mexico City, Mexico, Vice-Chairman
Otto C. Brantigan, M.D., Baltimore, Maryland, Secretary
Jose Ignacio Baldo, M.D., Caracas, Venezuela
Edward H. Robitzek, M.D., New York, New York

NEW AMERICAN MEDICAL DIRECTORY

Preparations are now being made to publish the new, Eighteenth Edition of the *American Medical Directory*. The last edition of the Directory was issued late in 1942. Since that time, it has been impossible to publish a new edition because of wartime restrictions and the shortage of paper and labor.

About November 15, a directory card will be mailed to every physician in the United States, its dependencies, and Canada, requesting information to be used in compiling the new Directory. Physicians receiving an information card should fill it out and return it promptly whether or not any change has occurred in any of the points on which information is requested. It is urged that those physicians also fill out the right half of the card, which information will be used exclusively for statistical purposes. Even if a physician has sent in similar information recently, mail the card promptly to insure the accurate listing of his name and address. There is no charge for publishing the data nor are physicians obligated in any way.

Should any physician fail to receive one of these Directory Information cards by December 1, he should write at once to the Directory Department of the American Medical Association, requesting a duplicate card be mailed.

IMPORTANT NOTICE: The cards being mailed by the American Medical Association do not contain a symbol to designate the physicians specializing in Diseases of the Chest. Physicians who wish to be classified in the American Medical Directory as specialists in Diseases of the Chest should write the American Medical Association, 535 North Dearborn St., Chicago, Ill., requesting that the symbol "DC" be used in their listings.

REPORT OF THE COMMITTEE ON CHEMOTHERAPY AND ANTIBIOTICS

Streptomycin is the most valuable and encouraging agent yet to be developed for the treatment of tuberculosis. After extensive animal experimentation and observation of its clinical use in several thousand patients, certain definite statements may be made. While much clinical investigation remains to be done, streptomycin may now be accepted as a valuable and at times necessary adjunct in the treatment of certain types of tuberculosis.

The optimum daily dose, frequency of administration, and duration of treatment with streptomycin for tuberculosis have not yet been fully established. The accumulated evidence indicates that for an adult 1 gram daily in divided doses given every 6 to 8 hours for 75 to 100 days may be sufficient in many cases. Some recent evidence indicates that a therapeutic dose may be as low as 0.25 gram given as infrequently as once a day. The drug is best given intramuscularly or by deep subcutaneous injection, but not intravenously, in solutions containing 100 to 250 mg. of streptomycin per c.c. Excretion is chiefly in the urine.

There is a certain toxicity, and undesirable side reactions may occur. No deaths have been reported resulting from the use of streptomycin. In general, the reactions are mild and even when severe are not sufficient to interfere with treatment of patients with progressive tuberculosis which is not amenable to other therapy. There is some question, because of these side reactions, whether streptomycin should be used in the treatment of patients who would respond satisfactorily to more conventional forms of therapy.

Disturbance of equilibrium due to loss of vestibular function is the most common of these reactions. There is a definite correlation between dosage of streptomycin and this vestibular disorder, which will occur in most of the cases receiving 2 grams or more per day for any long period of time; whereas, administration of 1 gram per day for the same period will cause no such disorder in more than half of the patients so treated.

Evidence of sensitization: chills, fever, nausea, and cutaneous rash—occurs occasionally within the first few weeks of treatment. The administration of the drug should be discontinued in such a case; and after a few days when the symptoms have subsided, the patient should be desensitized with daily doses commencing at 50 to 100 mg. and increased gradually to the customary therapeutic dose.

Streptomycin is essential in the treatment of tuberculous meningitis, miliary tuberculosis, tuberculosis of the larynx, trachea and bronchi, draining sinuses from tuberculous infection of bones and glands, and tuberculosis of the intestinal tract.

Caution and clinical judgment are required in the treatment of pulmonary tuberculosis with this agent. Not all cases are suitable for this treatment. Old chronic fibrocaseous lesions and thick-walled cavities are not usually affected much, if any, by this drug. Recently developed pneumonic and exudative lesions show the best response; recent and thin-walled cavities may close or show decrease in size.

The greatest use of streptomycin is as an adjunct to the more customary methods of treating pulmonary tuberculosis. The clinician should plan to combine streptomycin with collapse therapy, pulmonary resection, or whatever is indicated to produce a satisfactory result.

The factor which interferes most seriously with prolonged treatment with streptomycin is the development of streptomycin resistant strains of tubercle bacilli. This apparently occurs most frequently during the third or fourth month of treatment, and may limit the effectiveness of the treatment to that period. In many of these cases, subsequent treatment with streptomycin will be found to be ineffective. Some work is being done on combination of streptomycin with other substances which may aid in postponing the development of resistant strains.

It should be remembered that streptomycin cannot improve irreversible destructive tissue changes and, in general, cannot replace surgical treatment. It can and does widen the indications for surgical treatment by reducing toxicity, diminishing the tendency of the disease to spread, and causing resolution of many recent exudations. It can thus improve the patient's status to the point where surgical treatment may be done with greater safety and better end results.

Streptomycin is not a cure-all nor even a real cure. It will not replace sanatorium or hospital care nor substitute for accepted treatment in most cases. However, the time has come to state that its value has been established and that it has a definitely recognized place in the treatment of tuberculosis. It should be available to all patients in whom its use is a necessary or important adjunct to their recovery. For this reason an item covering the cost of this drug should be included in the annual budgets of all institutions established for, or undertaking the treatment of, tuberculosis.

Karl H. Pfuetze, M.D., F.C.C.P.,
Chairman, Subcommittee on Chemotherapy and Antibiotics

Edwin R. Levine, M.D., F.C.C.P.,
Chairman, Committee on Management and Treatment of Diseases of the Chest

Committee on Chemotherapy and Antibiotics:

Karl H. Pfuetze, M.D., Cannon Falls, Minnesota, Chairman
B. L. Freedlander, M.D., San Francisco, California, Vice-Chairman
Sumner S. Cohen, M.D., St. Louis Park, Minnesota, Secretary
Manuel Albertal, M.D., Buenos Aires, Argentina
Arnold Shamaskin, M.D., Hines, Illinois
Carl W. Tempel, Col., M.C., Denver, Colorado
John V. Thompson, M.D., Indianapolis, Indiana

REPORT OF COMMITTEE ON CHEST DISEASES IN PENAL AND MENTAL INSTITUTIONS*

Sufficient sporadic reports have stressed the important incidence and lack of uniform control programs of chest diseases in mental and penal institutions. Recent survey studies are emphasizing the high morbidity of tuberculosis. There appears, with exceptions, to be no persistent effort to repeat these analyses or afford modern therapy.

In some states a disproportionally high percentage of tuberculosis mortality is accounted for because of uncontrolled disease within governmental institutions.

Mental patients and penal inmates are forgotten and unwanted groups, but important foci of infection. These, the fellow employees and the communities to which discharges and parolees return have a right to expect more from the taxing and responsible agencies. Phthisis is still in the consumptive era in too many of the governmental confines.

Since these institutions are notoriously understaffed, the solution ultimately results in overcoming the problem of personnel. For example, one of the states has had a control program in effect over six years and at present has approximately 2000 mental patients with active tuberculosis. Lack of trained personnel has permitted only the simplest therapy.

An Advisory Committee of recognized chest specialists in one state has overcome administrative inertia and permitted an even flow of policy in spite of personnel changes.

Recommendations:

(1) The non-political appointment of advisory committees in each state deserves careful consideration.

(2) That the local chapters of the American College of Chest Physicians take an active interest in the problem of chest diseases in these special groups in their area.

(3) State Medical Societies should be informed of the "crying need" for adequate measures to combat this source of constant danger of infection for the population at large.

(4) The national organizations dealing with health problems should assume their part in the responsibility and guidance in carrying this problem to a successful termination. The combined efforts of all groups will be needed.

(5) In the present program of hospital expansion the need of separate units to care for these special groups must be emphasized. In planning these units architects should consult not only with psychiatrist and penologist but also with chest specialists.

(6) At least temporarily, the services of local specialists in diseases of the chest should be utilized wherever possible in lieu of permanent trained personnel.

Otto L. Bettag, M.D., Pontiac, Illinois, Chairman
B. B. Bagby, Jr., M.D., Martinsburg, West Virginia
G. C. Bellinger, M.D., Salem, Oregon
H. A. Burns, M.D., St. Paul, Minnesota
George M. Curtis, M.D., Columbus, Ohio
J. George Lang, M.D., New York, New York
A. A. Leonidoff, M.D., Poughkeepsie, New York

*Presented at the Annual Meeting, Board of Regents, American College of Chest Physicians, Atlantic City, New Jersey, June 5, 1947.

POSTGRADUATE COURSE IN DISEASES OF THE CHEST

Sixty-two physicians from 20 states, the District of Columbia, Canada, China, Cuba, Mexico and Spain, registered for the Postgraduate Course in Diseases of the Chest sponsored by the Council on Postgraduate Medical Education of the American College of Chest Physicians in Chicago, September 15th-20th, 1947. Letters received from the physicians who enrolled for the course indicated that this was by far the most outstanding course in diseases of the chest in which they have participated.

The faculty, under the chairmanship of Dr. Edwin R. Levine, Chief of the Chest Service at Michael Reese Hospital, Chicago, was recruited from New York City, Washington, D. C., Detroit, Denver, Minneapolis, Cannon Falls, Minnesota, Milwaukee, and Chicago. Each lecturer was selected because of his outstanding work in a particular phase of the specialty.

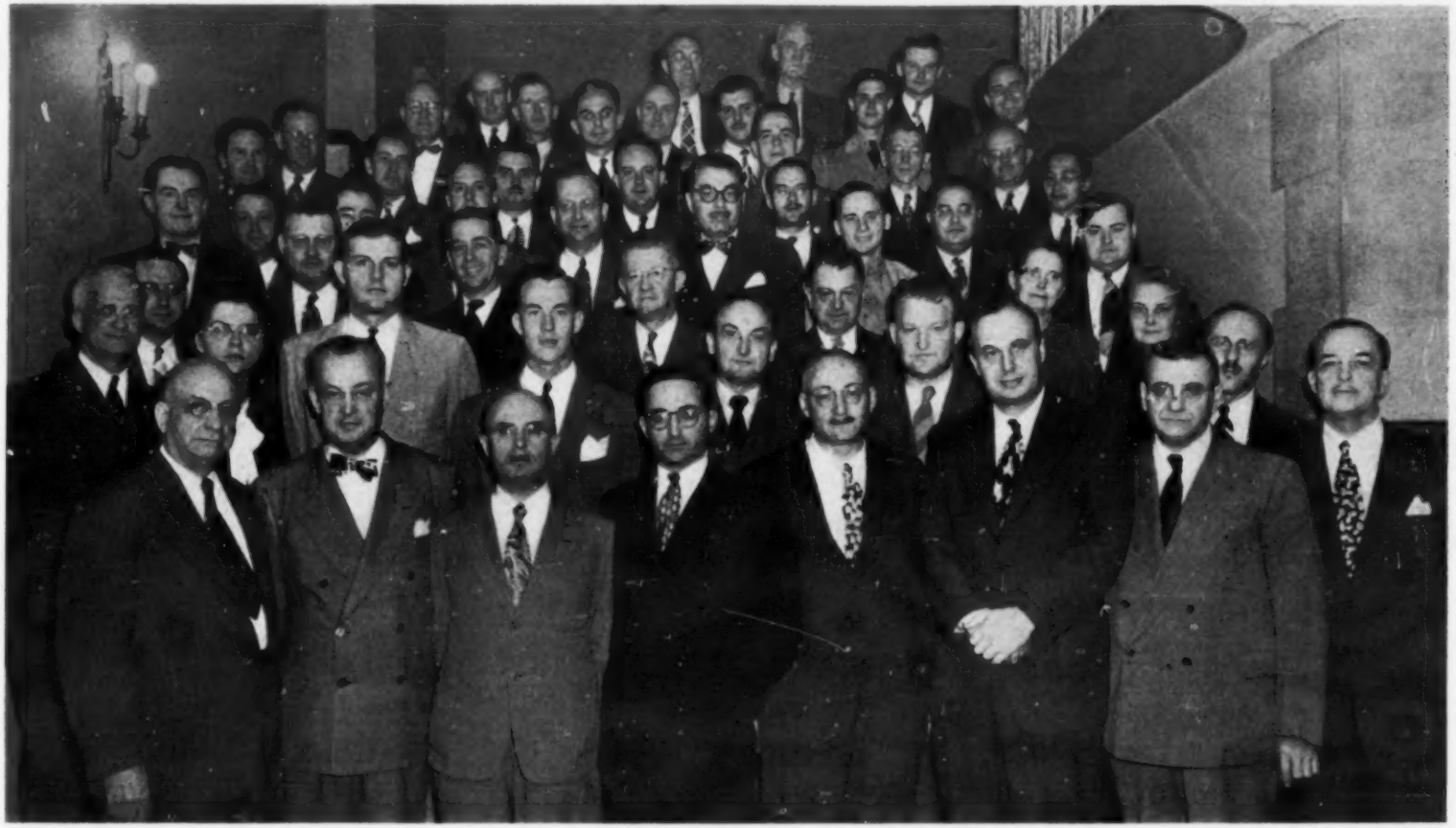
To close the week's program, a dinner was given by the Council on Postgraduate Medical Education of the College for the instructors and for the physicians who registered for the course. Dr. J. Winthrop Peabody, Washington, D. C., Chairman of the Council on Postgraduate Medical Education, presided. The following instructors attended the dinner: Dr. Andrew L. Banyai, Milwaukee, Wisconsin; and Dr. Paul H. Holinger, Dr. Minas Joannides, Dr. Edwin R. Levine, Dr. Arthur W. Newitt, and Dr. Leon Unger, Chicago, Illinois. Dr. Walter E. Vest of Huntington, West Virginia, was present and introduced.

Dr. George F. Grisinger, Charleston, West Virginia, class valedictorian, made the following remarks:

> "It is a pleasure and privilege for me, as Valedictorian for the 1947 Class of Physicians who participated in the Second Annual Postgraduate Course on Diseases of the Chest, in Chicago, to say a few words on behalf of my colleagues and myself to our instructors and to all others who have made this course so instructive and enjoyable to us.
>
> "We are deeply grateful to the physicians who have given so unselfishly of their time and energies to extend this very interesting course to us. We are happy to have had an opportunity to listen to these eminent teachers who have participated in this most enlightening course on diseases of the chest.
>
> "We particularly wish to thank Dr. Edwin R. Levine, Chairman of this splendid postgraduate course, for his untiring efforts in organizing and directing the course. We also wish to thank Mr. Murray Kornfeld, Executive Secretary of the College, and his staff, for the excellent arrangements in connection with the course. We wish, also, to express our appreciation to the management of the Municipal Tuberculosis Sanitarium, for their fine hospitality.
>
> "To each of the Instructors we desire to leave a sincere note of appreciation for the knowledge which has so generously been given to us in the Lecture Hall.
>
> "The Members of the Class, who have come from 20 States, the District of Columbia, Canada, China, Cuba, Mexico, and Spain, are deeply honored to be members of the Postgraduate Course of 1947.
>
> "It is our sincere hope that the course will be repeated annually, and that those who follow us will profit as much from the succeeding courses as we have profited from this one.
>
> "I again wish to thank you for your cordial hospitality and we shall remember the many happy days spent in Chicago."

SECOND ANNUAL POSTGRADUATE COURSE IN DISEASES OF THE CHEST, AMERICAN COLLEGE OF CHEST PHYSICIANS

September 15-20, 1947, Municipal Tuberculosis Sanitarium, Chicago, Illinois



Some of the physicians and instructors who participated in the Second Annual Postgraduate Course in Diseases of the Chest of the American College of Chest Physicians.

Following is a list of the physicians who registered for the course:

F. Kenneth Albrecht, M.D., Baltimore, Maryland
Pedro Alegria Garza, M.D., Mexico City, Mexico
W. H. Angerman, M.D., Massillon, Ohio
J. A. Baird, M.D., Fargo, North Dakota
Frank Barrera, M.D., Havana, Cuba
Lt. (jg) Robert L. Bauer, USN, Jacksonville, Florida
Capt. Frank A. Benchik, USA, Denver, Colorado
Lt. (jg) Charles A. Beskin, USN, Brooklyn, New York
John H. Bisbing, M.D., F.C.C.P., Reading, Pennsylvania
W. M. Blair, M.D., West Palm Beach, Florida
Mary C. Block, M.D., Santa Ana, California
William Block, M.D., Hartselle, Alabama
A. J. Bondurant, M.D., F.C.C.P., Springfield, Missouri
Lt. (jg) Carl D. Brannan, USN, Jacksonville, Florida
Walter B. Brown, M.D., San Fernando, California
Jaime Capo, M.D., F.C.C.P., Valencia, Spain
Capt. William Cernock, USA, El Paso, Texas
James W. Dimon, M.D., F.C.C.P., Utica, New York
Howard K. Edwards, M.D., F.C.C.P., Miami, Florida
Col. Paul Fancher, USA, Battle Creek, Michigan
Col. James O. Gillespie, USA, San Antonio, Texas
Andrew C. Goesl, M.D., Chicago, Illinois
Roy Goggans, M.D., Hillsboro, Texas
Col. Mack M. Green, USA (F.C.C.P.), San Francisco, California
George F. Grisinger, M.D., Charleston, West Virginia
William Grosfeld, M.D., F.C.C.P., Decatur, Alabama
Victor J. Hagen, M.D., Manitoba, Canada
Jesse W. Hofer, M.D., Chicago, Illinois
J. S. Hoffman, M.D., F.C.C.P., Kansas City, Missouri
Lt. (jg) Ralph A. Jessar, USN, Jacksonville, Florida
Lt. (jg) Seymour R. Kaplan, USN, Jacksonville, Florida
William W. Kearney, M.D., Oakdale, Iowa
Oliver Marcotte, M.D., F.C.C.P., Detroit, Michigan
Helen Marshall, M.D., Statesan, Wisconsin
Col. John McBride, USA, Scott Field, Illinois
Marshall L. McClung, M.D., Indianapolis, Indiana
Andrew Nady, M.D., F.C.C.P., Tucson, Arizona
A. H. Nejat, M.D., Woodhaven, Long Island, New York
Daniel N. Pickar, M.D., Louisville, Kentucky
Frank M. Quinn, M.D., Chicago, Illinois
Capt. Edgar Ricen, USN (F.C.C.P.), Corona, California
Joseph Rosenwasser, M.D., Mishawaka, Indiana
J. Rubin, M.D., F.C.C.P., Outremont, Canada
Paul T. Russell, M.D., Albany, Georgia
F. Fred Ruzicka, M.D., Baltimore, Maryland
Maurice Saibil, M.D., Montreal, Canada
Martin P. Sasko, M.D., Chicago, Illinois
Lt. (jg) Roy F. Saxon, USN, Great Lakes, Illinois
Maurice W. Sbertoli, M.D., Chicago, Illinois
William H. Shlaes, M.D., Chicago, Illinois
Richard C. Schneble, M.D., F.C.C.P., Dayton, Ohio
C. A. Slaughter, M.D., F.C.C.P., Houston, Texas
J. F. Spigler, M.D., F.C.C.P., Terre Haute, Indiana
A. J. Steiner, M.D., F.C.C.P., St. Louis, Missouri
Lloyd K. Swasey, M.D., F.C.C.P., Phoenix, Arizona
Y. K. Tseung, M.D., Honk Kong, China
Lt. Col. Joseph Vivas, USA, Washington, D. C.
Ruth Wells, M.D., F.C.C.P., Pasadena, California
Robert Wiener, M.D., Chicago, Illinois
Agatha Wilhelm, M.D., South Bend, Indiana
Charles Lee Williams, M.D., Chicago, Illinois
Comdr. Marion T. Yates, USN, Great Lakes, Illinois

College Chapter News

CALIFORNIA CHAPTER

Dr. Joseph L. Robinson, Los Angeles, President of the California Chapter of the College, has made the following committee appointments for the year 1947-1948:

Membership Committee:

John C. Sharp, M.D., Salinas, Chairman
William A. Cassidy, M.D., Livermore
James T. Harkness, M.D., Berkeley
Paul N. Smith, M.D., Olive View

Program Committee:

Jane Skillen, M.D., Olive View, Chairman
Cabot Brown, M.D., San Francisco
Gordon A. Diddy, M.D., Ahwahnee
William A. Kinney, M.D., Riverside
David T. Proctor, M.D., Pasadena

Nominating Committee:

Edward W. Hayes, M.D., Monrovia, Chairman
Forrest J. Bell, M.D., San Francisco
Jacob J. Singer, M.D., Beverly Hills

Postgraduate and Undergraduate Education Committee:

William L. Rogers, M.D., San Francisco, Chairman
Frank S. Dolley, M.D., Los Angeles
Seymour M. Farber, M.D., San Francisco
Edward W. Hayes, M.D., Monrovia

ILLINOIS CHAPTER

The Illinois Chapter of the College sponsored a scientific meeting at the Congress Hotel, Chicago, on Friday evening, November 7th. A dinner was held at the Congress Hotel at 6:30 and the following scientific program was presented at 8:00 p. m.:

The Place of Excision of the Lung in the Treatment of Pulmonary Tuberculosis

"Indications for Excision"
Edwin R. Levine, M.D., F.C.C.P., Chicago, Illinois.

"The Tuberculous Lesion of the Bronchus as it Affects Excision"
Paul H. Holinger, M.D., F.C.C.P., Chicago, Illinois.

"Problems of the Surgical Procedure"
Jerome R. Head, M.D., F.C.C.P., Chicago, Illinois.

"Care of the Patient Pre-operatively and Post-operatively"
George W. Holmes, M.D., Chicago, Illinois.

Discussion was opened by Minas Joannides, M.D., F.C.C.P., Chicago, Illinois, and many of those present participated.

INDIANA CHAPTER

The Indiana Chapter of the College met with the Anti-Tuberculosis Committee of the Indiana State Medical Association on October 28, during the 98th Annual Session of the Indiana State Medical Association held at the French Lick Springs Hotel, French Lick, Indiana. A luncheon meeting of the state and local county Anti-Tuberculosis Committees of the Indiana State Medical Society was held.

Following general discussion, an x-ray conference, sponsored by the Indiana Chapter of the College, was held, at which many interesting films were shown.

NEW ENGLAND STATES CHAPTER

The New England States Chapter of the College met at Boston, Massachusetts, on September 17. A scientific program was presented, which was very well attended, not only by many members of the College, but also by physicians from Spain, Belgium, Norway and Cuba.

The following officers were elected for the year 1947-1948:

U. E. Zambarano, M.D., Wallum Lake, Rhode Island, President
Moses J. Stone, M.D., Boston, Massachusetts, Vice-President
John B. Andosca, M.D., Mattapan, Mass., Secy.-Treas. (Re-elected)

NEW JERSEY CHAPTER

The New Jersey Chapter of the College held its Fall Clinical Program on October 21st at the Valley View Sanatorium, Paterson, New Jersey. Dr. Benjamin P. Potter, Jersey City, spoke on "Observation in Diagnosis and Treatment of Chest Diseases Commonly Encountered in the Practice of Medicine and Surgery." Interesting case reports were also presented by other chapter members. This meeting was a joint meeting of the Passaic County Medical Society and the New Jersey Chapter of the College.

The Board of Trustees of the State Medical Society of New Jersey during the year authorized the inclusion of a Session on Chest Diseases for its 1948 Annual Meeting to be held in Atlantic City, April 26-29. The meeting will take place at the Hotel Haddon-Hall. Dr. John E. Runnels, Scotch Plains, was appointed Chairman of the Session, and Dr. Homer H. Cherry, Paterson, was appointed Secretary.

The chapter reported that an active Membership Committee, under the Chairmanship of Dr. Joseph A. Smith, Glen Gardner, has resulted in an increase in the chapter membership.

PENNSYLVANIA CHAPTER

At the business meeting of the Pennsylvania Chapter of the College which was held in Pittsburgh on September 18, the following officers were elected for the year 1947-1948:

Burgess Gordon, M.D., Philadelphia, President
Elmer Highberger, M.D., Greensburg, Vice-President
Edward Lebovitz, M.D., Pittsburgh, Secy.-Treas. (Re-elected)

PUERTO RICO CHAPTER

The Puerto Rico Chapter of the College met in Guayama on September 28th, at the office of Dr. Jaime Fuster who played host to the group. Dr. David E. Garcia, Hato Rey, Regent of the College for Puerto Rico, presented a very interesting paper on "Pulmonary Lesions in Schistosomiasis" which stimulated much discussion between the clinicians and the radiologists. Dr. Luis A. Passalacqua of Santurce, talked of his personal experiences with six cases of "Vagotomy with Transpleural Approach for Peptic Ulcer" and Dr. Manuel Guzman-Rodriguez, Santurce, discussed the x-ray aspects of the problem.

A Board of Examiners was elected by the chapter at their meeting which will start functioning in January, 1948. The Board members elected are as follows:

Manuel Guzman-Rodriguez, M.D., F.C.C.P., Santurce
Jose Soto Ramos, M.D., F.C.C.P., Rio Piedras
Felix M. Reyes, M.D., F.C.C.P., Bayamon
Juan H. Font, M.D., F.C.C.P., San Juan
Jaime F. Pou, M.D., F.C.C.P., Hato Rey

ROCKY MOUNTAIN CHAPTER

At the annual meeting of the Rocky Mountain Chapter of the College, held September 16, at the Shirley Savoy Hotel, Denver, Colorado, the following officers were elected for the year 1947-1948:

Lorenz W. Frank, M.D., Denver, Colorado, President
J. E. J. Harris, M.D., Albuquerque, New Mexico, First Vice-President
R. G. Rigby, M.D., Salt Lake City, Utah, Second Vice-President
W. B. Yegge, M.D., Denver, Colorado, Secy.-Treas. (Re-elected)

TEXAS CHAPTER

Dr. Charles J. Koerth, Kerrville, Secretary-Treasurer of the Texas Chapter of the College, announces that Dr. H. Frank Carman, Dallas, President of the Chapter, has appointed the following committees for the year 1947-1948:

Medical Education Committee:

John Chapman, M.D., Dallas, Chairman
Howard T. Barkley, M.D., Houston
George S. McReynolds Jr., M.D., Galveston

Program Committee:

Charles J. Koerth, M.D., Kerrville, Chairman
Michael A. Cunningham, M.D., Beaumont
Cuthbert B. Young, M.D., Tyler

Public Relations Committee:

Walter C. Brown, M.D., Corpus Christi, Chairman
Ernest E. Holt, M.D., College Station
Ralph H. Homan, M.D., El Paso

Membership Committee:

William W. Coulter Jr., M.D., McAllen, Chairman
Wayne A. Reser, M.D., Wichita Falls
Hubert T. Ivey, M.D., Legion

Nominating Committee:

Charles M. Hendricks, M.D., El Paso, Chairman
Sim Hulsey, M.D., Ft. Worth
Howard E. Smith, M.D., Austin

Arrangements Committee (Houston Meeting):

J. Emerson Dailey, M.D., Houston, Chairman
Clarence R. Bruhl, M.D., Houston
Walter J. Stork, M.D., Houston

WISCONSIN CHAPTER

The Wisconsin Chapter of the College had a very successful meeting in Milwaukee on October 5, with more than 100 members and guests attending. New officers were elected for the Chapter for the ensuing year. They are as follows:

Ethan B. Pfefferkorn, M.D., Oshkosh, President
George H. Jurgens, M.D., Milwaukee, Vice-President
Leon H. Hirsh, M.D., Milwaukee, Secretary-Treasurer (Re-elected)

Dr. Ethan B. Pfefferkorn, President of the Chapter, has announced the following committee appointments for the year 1947-1948:

Program Committee:

Andrew L. Banyai, M.D., Milwaukee, Chairman
Earl E. Carpenter, M.D., Superior
David D. Feld, M.D., Milwaukee

General Arrangements Committee:

Douglas A. Gutheil, M.D., Milwaukee, Chairman
John C. Dundee, M.D., Waukesha
Oscar C. Heyer, M.D., Janesville
Karl E. Kimber, M.D., Waukesha
Louise G. Nezwarski, M.D., Hawthorne
Valentine O'Malley, M.D., Milwaukee

Educational Committee:

Herbert H. Christensen, M.D., Wausau, Chairman
William T. Clark, M.D., Janesville
Karl E. Kassowitz, M.D., Milwaukee
Thomas O. Nuzum, M.D., Janesville
Paul E. Pifer, M.D., Kenosha
Leonard L. Sanford, M.D., Hillsboro
John K. Shumate, M.D., Madison

Publicity and Reception Committee:

Mischa Lustok, M.D., Milwaukee, Chairman
Harry Barrell, M.D., Waukesha
John P. Fetherston, M.D., Milwaukee
William B. Ford, M.D., Milwaukee
Emil Rothstein, M.D., Wood

Membership Committee:

Leon H. Hirsh, M.D., Milwaukee, Chairman
Laurie L. Allen, M.D., Milwaukee
Raymond H. Evers, M.D., Madison
Isabelle T. Gadzikowski, M.D., Milwaukee
Thomas C. Nuzum, M.D., Janesville
Marres H. Wirig, M.D., Madison

Scientific Arrangements Committee:

George H. Jurgens, M.D., Milwaukee, Chairman
Philip P. Feingold, M.D., Milwaukee
Esther W. Goldberger, M.D., Milwaukee
Stanley R. Szymanski, M.D., Wood

X-Ray Conference Committee:

Richard P. Jahn, M.D., Milwaukee, Chairman
Henry A. Anderson, M.D., Stevens Point
William T. Clark, M.D., Janesville
John W. Connell, M.D., Fond du Lac
Leonard W. Moody, M.D., Bayfield

Nominating Committee:

Carl O. Schaefer, M.D., Racine, Chairman
Andrew L. Banyai, M.D., Milwaukee
Alfred A. Busse, M.D., Jefferson

CENTRAL AMERICAN CHAPTER TO BE ORGANIZED

The Second Central American Congress on Tuberculosis will be held in San Salvador, C. A., November 13-15, 1947. The following subjects are to be discussed:

"Resultados tardios de los distintos tratamento de la tuberculosis pulmonar"

"Tratamiento de las tuberculosis precoz"

"Tuberculosis y embarazo"

The following members of the American College of Chest Physicians will participate in the discussions:

Amadeo V. Mastellari, M.D., F.C.C.P., Panama City, Republic of Panama, Regent of the College for Central America.

Augustin A. Sosa, M.D., F.C.C.P., Panama City, Republic of Panama, Governor of the College for the Republic of Panama.

Enrico Coronado Iturbide, M.D., F.C.C.P., Guatemala City, Guatemala, Governor of the College for Guatemala.

Raul Blanco Cervantes, M.D., F.C.C.P., Provincia Cartago, Costa Rica, Governor of the College for Costa Rica.

Rene Vargas L., M.D., F.C.C.P., Managua, Nicaragua, Governor of the College for Nicaragua.

Carlos Gonzalez B., M.D., F.C.C.P., San Salvador, El Salvador, Governor of the College for El Salvador.

Arturo Blanco Solis, M.D., F.C.C.P., San Jose, Costa Rico.

Maximo V. Carrizo, M.D., F.C.C.P., Colon, Republic of Panama.

Plans are being formulated to organize the Central American Chapter of the College at this Congress.

College News Notes

Evarts A. Graham, M.D., F.C.C.P., St. Louis, Missouri, was presented with the Lister Medal by the Royal College of Surgeons in London on September 22nd.

Karl H. Pfuetze, M.D., F.C.C.P., Cannon Falls, Minnesota, has received a grant of $4,182 for research in streptomycin in tuberculosis, under the Grants-in-Aid Program of the National Institute of Health Research. G. R. Meneely, M.D., F.C.C.P., Nashville, Tennessee, received a grant from the same source in the amount of $40,581 for experimental study of myocardial infarction.

M. D. Bonner, M.D., F.C.C.P., Jamestown, North Carolina, presented a paper on "Bronchiectasis Clinic" at the Tri-State Medical Association of the Carolinas and Virginia's Forty-Eighth Annual Meeting, Greensboro, North Carolina, March 3rd and 4th.

Edgar W. Davis, M.D., F.C.C.P., Washington, D. C., and David Salkin, M.D., F.C.C.P., Hopemont, West Virginia, have had their paper entitled "Intrathoracic Gastric Cysts" published in the September 27th issue of the Journal of the American Medical Association.

Osler A. Abbott, M.D., F.C.C.P., Atlanta, Georgia, presented two papers at the meeting of the Medical Association of Georgia at Augusta, April 23rd. The titles of the papers were "Experiences with a New Method for the Control of Intrathoracic Aneurysms" and "The Clinical Significance of Hemoptysis: A Study of 1316 Patients with Chest Disease."

Ovidio Garcia Rosell, M.D., F.C.C.P., Lima, Peru, Governor of the College for Peru, presided at a special meeting of the Federacion Medica Peruana honoring Dr. Morris Fishbein, Editor of the Journal of the American Medical Association. The meeting was held in Lima on August 6th.

The following College members lectured in the Fall Graduate Instructional Course in Allergy given by the American College of Allergists under the auspices of the University of Cincinnati College of Medicine, at Cincinnati, Ohio, on November 3rd to 8th: Fred W. Wittich, M.D., Minneapolis, Minnesota; Bret Ratner, M.D., New York, N. Y.; J. Warrick Thomas, M.D., Richmond, Virginia; Ethan Allan Brown, M.D., Boston, Massachusetts; and Orval R. Withers, M.D., Kansas City, Kansas.

The following Fellows of the American College of Chest Physicians presented papers at the 12th Assembly of the United States Chapter of the International College of Surgeons which was held in Chicago, September 28th through October 4th: William F. Rienhoff Jr., M.D.; Paul H. Holinger, M.D.; Albert H. Andrews Jr., M.D.; and Chevalier L. Jackson, M.D. The following Fellows of the College had movies presented on the motion picture program for the meeting: Richard H. Overholt, M.D.; Edgar Davis, M.D.; and John Roberts Phillips, M.D.

Herman E. Hilleboe, M.D., F.C.C.P., Albany, New York, New York State Commissioner of Health, spoke at the Tuberculosis Institute in Rochester, New York on September 19th, at a meeting sponsored by various local agencies. Dr. Hilleboe reviewed the newer advances in the treatment of tuberculosis and their relation to the over-all control program.

Maurice G. Buckles, M.D., F.C.C.P., formerly of Louisville, Kentucky, has accepted a position on the faculty of the Ohio State University Medical School, department of chest surgery, and has opened private offices in Columbus, Ohio.

Brian A. Barlow, M.D., F.C.C.P., Dermott, Arkansas, was elected President of the Arkansas Tuberculosis Association during the annual meeting of the Association in Little Rock on September 10th.

Karl H. Pfuetze, M.D., F.C.C.P., Cannon Falls, Minnesota, visited Missouri on the 23rd and 24th of October. He talked at the Post-Graduate Assembly at the University of Kansas on Thursday, October 23rd, and later in the afternoon took a plane to south Missouri where he addressed the meeting of the Eighth District Council Meeting of the Missouri State Medical Association. Dr. Pfuetze's subject was "The Use of Streptomycin in Tuberculosis." On Friday the 24th, he talked to the staff members of the U. S. Veterans Administration Hospital at O'Reilly Hospital, Springfield, Missouri.

Joseph C. Placak, M.D., F.C.C.P., Cleveland, Ohio, Chairman of the Board of Regents of the College, was given the Distinguished Service Award by the Community Chest Organization of Cleveland for tireless and effective service in the prevention and cure of tuberculosis. In presenting the Award to Dr. Placak, Mr. Hal H. Griswold, Campaign Chairman of the Community Chest, stated that Dr. Placak had given unselfishly of his services to the community for a period of more than 40 years, and that in recognition of these fine services, he was elected as the recipient of this Award.
Cleveland Plain Dealer, October 22, 1947.

Otto L. Bettag, M.D., F.C.C.P., Pontiac, Illinois, Chairman of the Committee on Chest Diseases in Penal and Mental Institutions of the College, has announced that a meeting of the committee took place in Columbus, Ohio, November 14, 15 and 16.

FIRST ARGENTINE CONGRESS ON BRONCHOESOPHAGOLOGY

The First Argentine Congress on Bronchoesophagology will take place in Cordoba (Alta Gracia), Argentina on December 12 and 13, 1947. This Congress has been organized by the Argentine Society of Bronchoesophagology and the Society of Otorhinolaryngology of Cordoba. Drs. Chevalier Jackson and Chevalier L. Jackson of Philadelphia, Pennsylvania, are Honorary Presidents of the Congress, and Dr. Alvaro Bence, Buenos Aires, Argentina, is President of the Executive Committee.

The programs for the two days of the Congress are as follows:

December 12th:

"Indicaciones Diagnósticas y Terapéuticas en Broncología"
Alvaro Bence, M.D.

Discussant: Juan Manuel Tato, M.D.

"Indicaciones Diagnósticas y Terapéuticas en Esofagología"
Jose Ameriso, M.D.

Discussant: Antonio Carrascosa, M.D.

December 13th:

"Enseñanza Universitaria de la Broncoesofagología"
Roger Lanza Castelli, M.D.

Discussant: Tomas de Villafane-Lastra, M.D., F.C.C.P.

"Servicios Hospitalarios de Endoscopia Peroral"
Oscar A. Vaccarezza, M.D., F.C.C.P.

Discussant: Lazaro Langer, M.D.

"Endoscopia Peroral en el Niño"
Abelardo Irigoyen Freire, M.D.

Discussant: Emilio E. Tolosa, M.D.

Dr. Chevalier L. Jackson, Chairman of the Council on Pan American Affairs of the College, will be guest of honor at the Congress in Cordoba and while in Argentina he will give two courses in bronchoesophagology. One course will be given in Buenos Aires, December 1-7, for the physicians in that city and in the Province of Buenos Aires, and the other will be given in Cordoba, December 8-14, for the physicians in Cordoba and the interior of the Republic.

1947 EDITION OF PARERGON

Parergon (work by the side of work) is Mead Johnson & Company's picture book of artistic works by physicians. The current edition is a book of 208 pages and shows 1100 examples of creative art by contemporary physicians. This book is available without charge only to physicians upon request of Mead Johnson & Company, Evansville 21, Indiana, U. S. A.

TUBERCULOSIS CASE FINDING PROGRAM LAUNCHED BY THE NEW YORK STATE DEPARTMENT OF HEALTH

The New York State Department of Health has launched another phase of its intensified tuberculosis case finding program, according to announcement by Dr. Herman E. Hilleboe, Commissioner.

This new phase is one of promoting routine chest x-ray examinations of patients admitted to general hospitals having 7000 or more admissions per year through the loan of complete equipment for making small x-ray films. The objective of the program is to make an x-ray examination of each patient, 15 years of age and over, admitted to such a general hospital so that previously unknown cases of tuberculosis may be found. The chest x-ray examination of all hospital employees not previously examined and of all new employees is an important part of the program.

PACIFIC NORTHWEST DISTRICT CHAPTER

A meeting of the Pacific Northwest District Chapter of the College and the local chapter of the American Trudeau Society took place in Seattle, Washington, at the Medical and Dental Building Auditorium on October 30 and 31. The following program was presented:

Morning Session, October 30, 1947:

Symposium on the Use of Streptomycin in Tuberculosis

John Carswell, M.D., F.C.C.P., Vancouver, Washington.
William G. Lewis, M.D., F.C.C.P., Walla Walla, Washington.

"The Use of Streptomycin in British Columbia,"
William Hatfield, M.D., F.C.C.P., Vancouver, B. C.

"Miliary and Meningeal Tuberculosis Treated by Streptomycin. Case Report," John E. Tuhy, M.D., Portland, Oregon.

Discussion: Ellison F. White, M.D., Seattle, Washington,
Grover C. Bellinger, M.D., F.C.C.P., Salem, Oregon,
Frank I. Terrill, M.D., F.C.C.P., Deer Lodge, Montana.

Afternoon Session, October 30, 1947:

Symposium on Pulmonary Resection in Tuberculosis

Fred J. Jarvis, M.D., Seattle, Washington.
John E. Tuhy, M.D., Portland, Oregon.

Discussion: Frank I. Terrill, M.D., F.C.C.P., Deer Lodge, Montana,
Elliott Harrison, M.D., F.C.C.P., Vancouver, B. C.

"Auxiliary Techniques in the X-ray Diagnosis of Certain Pulmonary Lesions," Donal R. Sparkman, M.D., Seattle, Washington.

Dinner, New Washington Hotel, October 30, 1947:

Presentation of the President of the Pacific Northwest District Chapter, American College of Chest Physicians.

X-Ray Conference.

Morning Session, October 31, 1947:

Symposium on Anoxemia

Frederick R. Becker, Ph.D., Department of Anatomy, University of Washington Medical School.
Daniel M. Green, M.D., Department of Medicine, University of Washington Medical School.
Loren G. Carlson, Ph.D., Department of Physiology, University of Washington Medical School.

Discussion: James Blackman, M.D., Seattle, Washington,
"Mechanics of Respiration."

Luncheon

Organizational Meeting of Local Chapter of the American Trudeau Society.

Afternoon Session, October 31, 1947:

"Cardiac Surgery,"
William S. Conklin, M.D., F.C.C.P., Portland, Oregon.

"Treatment of Thoracic Aortic Aneurysms by Cellophane Wrapping,"
J. Karl Poppe, M.D., F.C.C.P., Portland, Oregon.

1948 COLLEGE DIRECTORY

The 1948 College Directory has been placed in the mails. If you have not received your copy please notify the Executive Offices of the College in Chicago.

This is the seventh issue of the Directory of the American College of Chest Physicians. The Directory, consisting of 364 pages, contains the names of 2274 Fellows, Associate Fellows, and Associate Members of the College whose applications were approved prior to May 15, 1947. Supplementary listings of new members will be printed and distributed to the membership of the College from time to time. The present membership of the College is 2405.

Members in the Directory are listed from 47 states, the District of Columbia, Alaska, Hawaii, Puerto Rico, Argentina, Australia, Belgium, Bolivia, Brazil, Canada, Chile, China, Colombia, Costa Rica, Cuba, Dominican Republic, Ecuador, El Salvador, England, Greece, Guatemala, Haiti, India, Italy, Jamaica, Jugoslavia, Lebanon, Mexico, New Zealand, Nicaragua, Norway, Paraguay, Peru, Philippine Islands, Portugal, Republic of Panama, South Africa, Sweden, Switzerland, Uruguay and Venezuela. The 1943 Directory of the College contained the names of 1255 members. The 1948 Directory shows a substantial increase both in members and in the number of countries in which there are members of the College.

The Directory contains the By-Laws and Articles of Incorporation which were enacted at the Eighth Annual Meeting of the College held in Atlantic City, New Jersey, June 6-8, 1942. It lists the names of the past presidents, and of chapters with dates of organization.

The Board of Regents of the American College of Chest Physicians, under whose supervision this Directory has been compiled, trusts that the Directory will be of service to the members of the College.

Additional copies of the Directory are available at $5.00 per copy.

SCHOLAR IN MEDICAL SCIENCE PROGRAM ANNOUNCED BY MARKLE FOUNDATION

$250,000 Annually Available for Five-year Post-fellowship Grants beginning 1948-49

An opportunity to start a career in academic medicine is offered to young scientists with the necessary training to hold a regular faculty appointment and to conduct original research through a new program of "post-fellowship" grants, announced by the John and Mary R. Markle Foundation. The purpose of the program, according to John M. Russell, Executive Director of the Foundation, is to attract much needed talent to academic medicine by giving promising young scientists academic security and financial assistance for a period up to five years. The program will be conducted in cooperation with accredited medical schools in the United States and Canada. Grants of $25,000, payable to the cooperating school at the rate of $5,000 annually for a five-year period toward the support of each successful candidate or his research or both, will be available beginning with the academic year 1948-49. If the plan proves successful, the Foundation will appropriate a total of $1,250,000 to the schools by 1953.

Candidates will be recommended by medical schools and will be limited

to young men and women with a particularly strong interest in research and teaching in any of the clinical or pre-clinical sciences or in the sciences basic to medicine. They will have had training in some special field or combination of fields to qualify them to receive a regular faculty appointment and to conduct original research. The final choice will be made, on the basis of the school's recommendations, by regional committees appointed by the Foundation. The young scientists chosen will be known as "Scholars in Medical Science." No fixed number of Scholars will be appointed in any year, but it is expected that approximately fifty will receive appointments during the five-year period. For each Scholar, the school will determine salary and academic rank, encourage research by setting reasonable limits upon teaching and other non-research activities, provide laboratory facilities, and, if necessary make a financial contribution toward the support of his work.

The Scholar program places the emphasis on the personal qualities and scientific and teaching abilities of the men and women chosen, rather than upon particular research projects or teaching fields in which they may be interested. The program is the result of a survey of medical research and education, recently made by the Foundation, which shows that while there are scholarships and other forms of financial aid for the student in the course of his scientific training and while there are funds available to the scientist once his name is made, there are few sources of help at the beginning of the career of the man who chooses academic medicine.

A pamphlet covering the details of the plan has been sent to all deans of accredited medical schools, and persons interested in being considered as candidates are referred to them for further information.

Book Reviews

Diseases of the Chest: With Emphasis on X-Ray Diagnosis: By Eli H. Rubin, M.D., F.A.C.P., F.C.C.P., Attending Physician, Division of Pulmonary Diseases, Montefiore Hospital and Country Sanatorium, New York; Visiting Physician in Tuberculosis and Physician-in-Charge, Chest Clinic, Morrisania City Hospital, New York. 685 pages, with 355 illustrations (24 plates in color). Philadelphia and London: W. B. Saunders Company, 1947. Price $12.00.

A valuable contribution to the basic understanding of the diagnosis and treatment of diseases of the chest is presented by Eli H. Rubin, M.D., and Morris Rubin, M.D., of New York City. The authors state they are emphasizing roentgen diagnosis but they do not neglect disturbances in pathological physiology which, in the last analysis, are also essential in diagnosis and treatment. Important contributions from the literature, as well as personal experiences, are included so that the book has definite merit for the chest specialist as well as the general practitioner and internist. Excellent illustrations and roentgen reproductions are used profusely and add much to the more complete understanding of the topics presented.

The contents of the book are divided into six major sections. The initial section deals with anatomic considerations and the technical aspects of chest roentgenography including fluoroscopy, usual roentgen techniques, tomography, angiography, kymography, fluorography and mass roentgenography. The technique and importance of bronchospiro-

metry in determining the respiratory function of each lung separately is outlined carefully. The advantages of roentgen diagnosis as compared to physical examination are stressed.

The second section deals with acute and chronic pneumonias. The indications and contraindications of penicillin and the sulfonamides are tabulated adequately and the possible dangers of the sulfonamides are emphasized. The discussion of suppurative pneumonias or lung abscesses is excellent and proper emphasis is placed on the relation between medical and surgical care.

The third section of the book is concerned with pulmonary tuberculosis. The authors feel that the primary infection with the tubercle bacillus leads to early generalization and the development of scattered hematogenous foci. The importance of routine mass roentgen surveys is stressed and the authors point out that about one per cent of clinically significant tuberculosis will be found in unselected segments of the population. Of these, about one-third will have active pulmonary tuberculosis and two-thirds arrested or inactive disease. In those patients who have developed symptoms, the onset will be insidious, catarrhal, pleural, hemoptoic or pneumonic.

The important laboratory procedures recommended for the diagnosis of pulmonary tuberculosis are frequent examinations of sputum or gastric contents for tubercle bacilli, the erythrocyte sedimentation rate and the tuberculin test. The accepted methods of examination for the recovery of tubercle bacilli are given in some detail. The authors agree that the presence of the organism is the only infallible evidence of the existence of pulmonary tuberculosis.

The decreasing incidence of complications in pulmonary tuberculosis is pointed out in the next chapter. This is thought to be due to earlier diagnosis and improved methods of treatment. The authors quote illustrative figures from the New York Municipal Sanatorium, Otisville, New York, where in 1914, 25.6 per cent of the patients examined had laryngeal tuberculosis as a complication while in 1941, the incidence had decreased to 3.6 per cent. The handling of nontuberculous conditions such as amyloidosis, syphilis, heart disease, diabetes and pregnancy when these coexist with tuberculosis is described.

The medical and surgical treatment of pulmonary tuberculosis is discussed in the following chapter. The nonspecific and specific therapeutic measures are described and pneumothorax is outlined in some detail. The authors mention BCG briefly and conclude its importance in this country is not great due to the continuing decline in the prevalence of tuberculosis. There is just a brief mention of streptomycin which is undoubtedly due to the completion of the book prior to the reports of its clinical use in tuberculosis. The surgical measures considered valuable include intrapleural and extrapleural pneumonolysis, diaphragmatic paralysis, pneumoperitoneum, thoracoplasty, cavity drainage and lung resection.

The fourth major section deals with other important diseases of the lungs and bronchi including the pneumonoconioses and other occupational hazards, bronchial obstruction and bronchial asthma. The authors outline the steps necessary in making an early diagnosis of bronchiogenic carcinoma and advise the attending physician to follow the principle that any obscure lung disease occurring in an individual of cancer age is carcinoma unless proved otherwise. The increasing incidence of bronchiogenic carcinoma is ascribed to an aging population, improved

methods of diagnosis and a greater accuracy in histologic differentiation. The absence of pathognomonic symptoms and the important diagnostic measures are properly emphasized.

The fifth section of the book is devoted to diseases of the mediastinum, diaphragm, pleura and related structures, including heart-lung disease. Mediastinal abnormalities including emphysema, infections, tumors and lymph nodes are described fully. The chapter on the diagnosis of pleural effusions will be found valuable by the physician. The authors point out the difficulties encountered in making a definite diagnosis of the underlying disease in primary pleural effusion.

The concluding section of the book covers the principles of surgical treatment, emphasizing preoperative and postoperative care. The differences, from a surgical standpoint, between the individual with acute suppurative disease of recent origin and the chronically ill patient with chronic pulmonary suppurations, bronchiectasis, neoplasms and tuberculosis are stressed from the standpoint of preoperative medical care and the frequency of postoperative complications. Bronchoscopic examination to determine the operability of neoplasms, the exact localization of abscesses and the presence or absence of endobronchial tuberculosis prior to the induction of pneumothorax is recommended. Brief discussions of surgical technique are included, as the authors state, in order to enable the student to appreciate what the thoracic surgeon is doing. The final chapter deals with chest emergencies and includes treatment of traumatic hemothorax, traumatic and tension pneumothorax, crushing chest wounds and thoraco-abdominal injuries.

The authors have included an excellent bibliography for each subject discussed and the contents are well indexed. There are few typographical errors. There is one error of some importance on page 81 where the dosage of sulfadiazene is given by the authors as approximately 0.5 gm. per pound where 0.05 gm. per pound is probably meant. This book can be recommended to all physicians interested in diseases of the chest.

Sumner S. Cohen, M.D.

Review of Dr. Francis Kovats' book *"Pulmonar Tuberculosis,"* Budapest, 1945, by A. Rottmann in the Wiener medizinische Wochenschrift, No. 4, 1947.

This book of the well-known Hungarian research worker and Professor of Diseases of the Lung, University of Budapest, is one of the best contributions which have been published recently in this particular field. It is particularly welcome today, for it points toward new therapeutic measures against this tremendously increased endemic disease. This book is an outstanding one in this respect. As stated by the author in the introduction, the work is based predominantly on empiricism, and on the basis of his wide experience, he offers important and enlightening data relative to theoretical concepts pertaining to this subject. A close portrayal of the evolutional changes of tuberculosis gives a clear understanding of its problems. Very properly, the author emphasizes the extreme importance of x-ray in the diagnosis of tuberculosis and accordingly there is an excellent selection and reproduction of 223 roentgenograms and diagrams which bring to light the standard as well as the more unusual diagnostic features of this disease. He emphasizes the importance of proper training and experience in roentgenologic technique and diag-

nosis as indispensable for clinical work. The clinical classification of the disease is presented in a concise and easily understandable manner. With regard to treatment, adequate emphasis is placed on various forms of collapse therapy. A masterful survey of the subject, together with a clear and penetrating presentation of the material render this book a fundamental source of information for chest physicians as well as for the general practitioner.

Andrew L. Banyai, M.D.

Obituaries

DRUE KING

1887 - 1947

Dr. Drue King was born in Augusta, Georgia, on March 16, 1887, the youngest of 14 children. He attended public school in Atlanta and then in Boston, and completed high school in Boston. He graduated from Tufts Medical College in 1914 and interned at John Andrew Memorial Hospital in Tuskegee, Alabama. At the expiration of his internship he returned to Augusta where he entered into private practice. In 1925 he was appointed to the position of Ward Surgeon at the Veterans Hospital in Tuskegee, Alabama. In 1927 Dr. King studied at the Laennec Clinic in Paris, France.

Dr. King was a Certified tuberculosis specialist and Senior Physician in point of Service on the staff of the Veterans Hospital, Tuskegee, Alabama. He served the Veterans Administration faithfully for 24 consecutive years.

On April 17, 1947, Dr. King suffered a cerebral hemorrhage. He died on April 20. He leaves to mourn his loss, a wife and two sons, the elder, Drue Jr., a recent graduate of Harvard College and now a medical student at Tufts Medical College, and the younger, Abram Oliver, a pre-medical student at Boston College.

Roy A. Wolford, M.D., Governor for the Veterans Administration.

LEE T. FERRELL

1879-1947

Dr. Lee T. Ferrell was born October 23, 1879, at Hazelhurst, Mississippi. He was graduated from the Medical School of the University of Tennessee in 1911 and engaged in private practice in Murphy, Mississippi from 1911 to 1918. He served with the Medical Corps of the United States Army during World War I and entered the Veterans Administration service in December, 1920. He died on February 3, 1947, and is survived by his wife, Mollie Clancy Ferrell, and a son, Lieutenant Colonel Lee T. Ferrell Jr., who is in the United States Army Medical Corps, Washington, D. C.

Dr. Ferrell was for a number of years on the staff of the Veterans Hospital at Albuquerque, New Mexico, and was highly respected by his associates and the medical fraternity in Albuquerque.

Robert O. Brown, M.D., Governor for New Mexico.

JOHN DONNELLY

1878 - 1947

Dr. John Donnelly was born at Connellsville, Pennsylvania, in 1878. At the age of eight his family moved to Charlotte, in Mecklenburg County, North Carolina. He was an honor student in the Charlotte public schools and at the University of North Carolina, where he won the degree of Bachelor of Arts and Doctor of Medicine.

Dr. Donnelly first engaged in the practice of medicine in Hillsboro, North Carolina, where he remained for only two years. He returned to Charlotte and soon was appointed to the faculty of the North Carolina Medical College. He was especially interested in tuberculosis and chest diseases. He developed an active tuberculosis and chest clinic, and though the school was discontinued in a few years, he continued the chest clinics in the City Health Department. He volunteered for service in World War I, in which he served with fidelity and distinction. With this war over, Dr. Donnelly came home and again resumed his warfare against tuberculosis.

A few years later, largely through sentiment developed by Dr. Donnelly's unremitting labors, Mecklenburg County provided a Sanatorium for the care of it's tuberculous, and Dr. Donnelly was made it's first superintendent and medical director, in which capacity he served for ten years. On resigning from the Sanatorium, Dr. Donnelly did private practice in Charlotte in diseases of the chest, and supervised the chest clinic for the City Health Department.

Due to hypertensive cardiovascular disease, his practice was necessarily limited for the last two or three years of his life. However, he was very actively interested in all the proceedings of the College until his death on April 16, 1947.

M. D. Bonner, M.D., Governor for North Carolina.

WALTER H. WATTERSON

1875-1947

Dr. Walter H. Watterson was a pioneer in the field of tuberculosis work in Illinois. His interest in tuberculosis was prompted by the fact that he was a patient himself in the days before the modern conception of treatment. He began practice at the age of 26. At 29 he had to take time out and "Go West." Several years later he established a tent colony for the tuberculous at Waukegan, Illinois (1908). He later was chief of Tuberculosis Service at Oak Forest from 1914-1916. In 1917-1918 he was medical superintendent of the Chicago Municipal Tuberculosis Sanatorium. He saw army service in 1918-1919 and for fourteen years following he was medical director of Zace Sanatorium at Winfield, Illinois. An acute illness forced his retirement. For over a year he was unable to work but later returned to a very active private practice in which he was engaged when he succumbed to a coronary occlusion on June 17th at the age of 72 years at his home in LaGrange, Illinois.

Robert K. Campbell, M.D., Governor for Illinois.

IRA. D. NELSON

1878 - 1946

The death of Dr. Ira D. Nelson on October 6, 1946, at Albuquerque, New Mexico, closed the career of a highly respected and valued physician and worker in the field of public health, particularly in tuberculosis, in the United States Indian Service. Dr. Nelson was born near Keokuk, Iowa, on September 11, 1878, graduated from Iowa Medical College in 1904, and took postgraduate work at the Mayo Clinic and the University of Minnesota. He began a long and valued career in the United States Indian Service during World War I, serving at many Indian Agencies, and as Superintendent at the government sanatoria at Rapid City, South Dakota; Toledo, Iowa; and at Claremore, Oklahoma. His success in work with tuberculosis, and in building up hospitals, was recognized in his appointment as Executive Medical Officer for the United Pueblos Agency and as Physician in Charge of the United States Indian Service Tuberculosis Sanatorium at Albuquerque, New Mexico, as well as of four other hospitals in the Southwest, until failing health forced his retirement from active duties.

Dr. Nelson, who was a Fellow of the American Medical Association, as well as of the American College of Chest Physicians, is survived by his widow, now living in Wellington, Kansas, two daughters, and a son, the latter a Medical Officer in the Army of Occupation in Germany.

Robert O. Brown, M.D., *Governor for New Mexico.*

JAHARLAL GHOSH

1907 - 1946

We regret that reports of the death of Dr. Jaharlal Ghosh have been confirmed. Dr. Ghosh was born in Dinajpur, Bengal, India. He received his medical degree from the University of Calcutta in 1932, and took postgraduate work at the Carmichael Medical College, Calcutta, and the Brompton Hospital, London.

Dr. Ghosh entered private practice in Calcutta and was also Clinical Tutor in Medicine and Lecturer in Medical Anatomy and Physiology at the Carmichael Medical College there. He was a Fellow of the American College of Chest Physicians, and served as Governor of the College for India, and a member of the Indian Medical Association, the Royal College of Physicians, London, and the Royal College of Surgeons, England.

Ramon Viswanathan, M.D., Governor for India.

PUBLIC HEALTH SERVICE AWARDS FOR MEDICAL RESEARCH

Public Health Service awards for basic medical research have totalled $10,214,174 during the past twenty months. Six hundred twenty-nine scientists in 193 institutions shared in these grants. Broken down into major categories of medical research, the total amounts and total number of projects are as follows:

Antibiotics, 27 projects, $249,698; Bacteriology, 43 projects, $445,689; Biochemistry and Nutrition, 49 projects, $682,358; Cancer, 114 projects, $1,241,510; Cardiovascular, 31 projects, $703,187; Dental, 25 projects, $135,607; Gerontology, 11 projects, $213,172; Hematology, 33 projects, $498,833; Malaria, 21 projects, $543,208; Mental Health, 28 projects, $276,-127; Metabolism and Endocrinology, 30 projects, $267,359; Pathology, 21 projects, $176,545; Pharmacology, 30 projects, $313,779; Physiology, 52 projects, $676,836; Public Health Methods, 9 projects, $125,843; Radiobiology, 16 projects, $392,399; Rodent Control, 2 projects, $17,120; Sanitation, 25 projects, $202,545; Surgery, 16 projects, $159,910; Syphilis, 46 projects, $1,669,793; Tropical Diseases, 20 projects, $352,987; Tuberculosis, 19 projects, $360,113; Virus and Rickettsial, 31 projects, $509,556.

The George Washington University School of Medicine

A N N O U N C E S

THIRD ANNUAL SERIES OF INTENSIVE POSTGRADUATE COURSES

Thoracic Surgery and Endoscopy ..... March 1-5, 1948
Thoracic Medicine ..... March 8-12, 1948
Internal Medicine (including Thoracic Medicine) ..... March 1 - April 16, 1948

A DISTINGUISHED GUEST FACULTY WILL SUPPLEMENT THE LOCAL STAFF

Numerous other courses are also available. Registration in each course is limited. Veterans can use their benefits under the G. I. Bill of Rights. Hotel accomodations are available. For further details write:

Director of Postgraduate Instruction
The George Washington University School of Medicine
1335 H Street, N. W. — Washington 5, D. C.

Medical Service Bureau

In accordance with a resolution adopted by the Board of Regents of the College at their annual meeting held in Chicago on June 17, 1945, a Medical Service Bureau has been established at the Executive Offices of the College for the purpose of serving the members of the College being released from the armed forces.

The Bureau would appreciate receiving information from the medical superintendents of sanatoria regarding positions available at their institutions, together with full particulars as to the type of position and salary offered. Fellows of the College who are looking for assistants should send complete information to the Bureau.

Physicians being released from the armed forces who are seeking appointments and positions should send complete information to the Bureau regarding their training and the type of position desired.

Please direct all correspondence to the Medical Service Bureau, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

POSITIONS AVAILABLE

Assistant Resident Physician with some tuberculosis experience wanted for 120-bed bi-county institution. All phases of the diagnosis and treatment of tuberculosis are carried out including major surgery. California license required although not immediately. Furnished apartment is provided for single person or married couple. Please outline training and experience in first letter and include a recent snapshot. Salary starts at $425.00 per month. Write Director, Tulare-Kings Counties Joint Tuberculosis Hospital, Springville, California.

Assistant Medical Director wanted, 170-bed tuberculosis hospital. Completely equipped thoracic surgery department, x-ray, laboratory and rotating staff. Salary open. Please address Mahoning Tuberculosis Sanatorium, 4880 Kirk Rd., Youngstown 7, Ohio.

Resident physician for 500-bed hospital wanted, providing all facilities in connection with hospital care, out-patient service with every agency that is not being employed in the treatment of tuberculosis and other diseases of the chest. Compensation in accordance with previous experience. Maintenance for the physicians, no provision for families. Berthold S. Pollak Hospital for Chest Diseases. B. S. Pollak, M.D., Medical Director, 100 Clifton Place, Jersey City 4, New Jersey.

Tuberculosis residency available; salary $250 to $350 per month for single man; must be graduate of American medical school. For further information please address Box 173A, American College of Chest Physicians, 500 N. Dearborn Street, Chicago 10, Illinois.

Young physician wanted, single, having served his internship and preferably having had a little experience in tuberculosis; 180-bed sanatorium; good opportunity for the right man; salary $2400 per year and maintenance. Dr. W. A. Bridges, Supt. and Med. Dir., Endowood Sanatorium, Towson 4, Maryland.

POSITIONS WANTED

Physician soon to be released from service desires residency in chest diseases. Experience includes an approved rotating internship, one year in an approved tuberculosis sanatorium, and at present on large tuberculosis service in Navy. Age 27, married, one child. For further information please address Box 234A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Well trained chest surgeon, bronchoscopist, qualified for the American Board of Surgery, interested in an association with established clinic, or a position in hospital or sanatorium with opportunity for private practice. For further information please address Box 235A, American College of Chest Physicians, 500 N. Dearborn Street, Chicago 10, Illinois.

Well trained chest surgeon desires position in an institution or an association with another chest surgeon. For further information please address Box 236A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Fellow, American College of Chest Physicians, chest surgeon, bronchoscopist, well trained, formerly member of university teaching staff, is interested in position with hospital or sanatorium with opportunity for private practice or in association with well established clinic. For further information please address Box 237A, American College of Chest Physicians, 500 N. Dearborn Street, Chicago 10, Illinois.

Reprints, Books and Reports Received.

1. Radiology, Section XIV, of Excerpta Medica, Vol. I, No. 1, June 1947.
2. Epidemiologia de la Tuberculosis en Chile, Dr. Benjamin Viel, 1946.
3. "Pneumothorax et Activite Locale du Foyer Tuberculeux Pu'monaire," Revue Belge de la Tuberculose, September-October, 1938.
4. "La Richesse Des B.K. en Granulations Gramophiles au Cours de la Tuberculose Pulmonaire," Revue de la Tuberculose, April 1938.
5. "Tomografia del Torax en Cortes Metamericos," Anales de La Catedra de Patologia Y Clinica de La Tuberculosis, June 1945.
6. "Reactions to Penicillin," by Sidney Dressler, M.D., and Ralph E. Dwork, M.D., Journal of American Medical Association, March 22, 1947.
7. "Tuberculosis Case Finding," by John A. Foley, M.D., and John B. Andosca, M.D., New England Journal of Medicine, December 5, 1946.
8. "The Significance of the Retracted and Curved Horizontal Fissure of the Lungs," by Ralph E. Dwork, M.D., American Journal of Roentgenology and Radium Therapy, March 1947.
9. Annual Report for the Year 1946, Erie County Tuberculosis Hospital, Erie, Pa.
10. Annual Report for Year Ending November 30, 1946, Du Page County Tuberculosis Sanatorium Board.
11. "El Embolismo Pulmonar," by Egidio S. Mazzei, M.D., Diego Taylor Gorostiaga, M.D., Elyeser Magalhaes, M.D., El Ateneo, 1947.
12. "Tuberculosis Case Finding," by Carl C. Birkelo, M.D., W. Edward Chamberlain, M.D., Paul S. Phelps, M.D., Percy E. Schools, M.D., David Zacks, M.D., Jacob Yerushalmy, M.D., Journal of American Medical Association, February 8, 1947.
13. "Prospects for Prevention of Chronic Bronchitis and Bronchiectasis," by Walter Finke, M.D., Scientific Exhibits of 5th International Congress of Pediatrics, July 14-17, 1947, New York, New York.
14. "Notes Sur une Manifestation Connexe de la Primo-Infection: L'Erytheme Noueux, Ses Rapports Chronologiques Avec les Stades de la Maladie Tuberculeuse," by Rene Jeanneret, M.D., Journal Medical de Leysin, February 1943.
15. "Tomographie et Pratique Phtisiologique," Radiologia Clinica, March 1944.
16. "A Propos du Pneumothorax Mixte, Intra et Extra-Pleural Associe," Revue Medicale, by Rene Jeanneret, M.D., and Rancois de Kovats, M.D., June 25, 1944.
17. "Kystes Gazeux du Poumon," by Rene Jeanneret, M.D., and Henri Mean, M.D., Revue Medicale de la Suisse Romande, November 25, 1940.
18. "A Propos des Ombres Radiologiques Fugaces," by Rene Jeanneret, M.D., and F. Fame, M.D., Revue de la Tuberculose, December 1933.
19. "Reprise Eloignee des Fonctions du Diaphragme Apres Phrenicectomie," by Rene Jeanneret, M.D., M. Ribert, M.D., and F. Fame, M.D., La Presse Medicale, May 9, 1934.
20. "La Pleuroscopie et la Section des Brides Pleurales dans le Pneumothorax Artificiel," by Rene Jeanneret, M.D., and F. Fame, M.D., Helvetica Medica Acta, Vol. 2, 1936.
21. "Indications et Resultats de la Phrenicoexerese dans la Tuberculose Pulmonaire de L'Adulte," by Morin, Cardis, and Michetti.
    "A Propos de la Phrenicectomie," by Piguet and Rene Jeanneret.
    "Communication des Resultats de 27 Phrenicectomies," by Rossell.
    "La Retraction Elective Dans La Phrenicoexerese," by Cardis and Joannette, Societe des Medecins de Leysin, July 4, 1929.
22. "Note Sur les Endometriomes," by Rene Jeanneret, M.D., Revue Medicale de La Suisse Romande, November 25, 1926.
23. Une Application Pratique de la Methode de Desensibilisation par les Cuti-Reactions Repetees," by Rene Jeanneret, M.D., Revue Medicale de la Suisse Romande, February 25, 1929.
24. "Deux Cas de Chorio-Epithelioma Malin Chez L'Homme," by Rene Jeanneret, M.D., Schweizerischen Medizinischen Wochenschrift, 1928.
25. "Frequence et Comportement de la Tuberculose Chez les Etudiants en Medecine des Universites Suisses," by Rene Jeanneret, M.D., Revue Suisse de la Tuberculose, Vol. II, 1945.
26. "Une Complication Rare de la Collapsotherapie Gazeuse," by Rene Jeanneret, M.D., Revue Suisse de Medecine, May 15, 1941.
27. "Note Sur la Pleuroscopie et les Sections D'Adherences," by Rene Jeanneret, M.D., and Andre Gilliard, M.D., Journal Suisse de Medecine, No. 6, 1942.
28. "La Pleuroscopie et la Section Des Adherences Pleurales Dans le Pneumothorax Artificiel," by Rene Jeanneret, Tuberculose Cours de Leysin, 1944.
29. "A propos de L'Aspiration Intra-Cavitaire des Cavernes Pulmonaires," by Rene Jeanneret, Journal Medical de Leysin, June 1943.
30. "Section D'Adherences et Collapsotherapie Gazeuse Efficace Chez un Diabetique, Tuberculeux-Pulmonaire," by Rene Jeanneret, M.D., Journal Suisse de Medecine, No. 23, 1940.
31. "A Propos de Procede D'Aspiration Intra-Cavitaire des Cavernes Pulmonaires Selon Monaldi," by Rene Jeanneret, M.D., and Gustave Joyet, Revue de la Tuberculose, July 1939.
32. "A Propos du Chorio-Epithelioma Malin Chez L'Homme," by Rene Jeanneret, M.D., Presented to the Faculty of Medicine of La Usanne University to obtain a Doctor of Medicine.
33. "Essai de Calssement des Adenopathies Tracheo - Bronchiques Tuberculeuses du

Jenne Adulte," by Rene Jeanneret, M.D., and F. Fame, M.D., Revue Medicale de la Suisse Romande.

34. "A Propos de la Localisation Radiologique des Adenopathies Tracheo-Bronchiques," by Rene Jeanneret, M.D., and F. Fame, M.D., Revue de la Tuberculose, February 1932.
35. "L'Image Radiologique de la Broncho-Pneumonie Grippale," by Rene Jeanneret, M.D., and F. Fame, M.D., Revue Medicale de la Suisse Romande, June 10, 1931.
36. "Note Sur L'Image Radiologique des Processus Pulmonaires Exsudatifs," by Rene Jeanneret, M.D., and F. Fame, M.D., Schweizerischen Medizinischen Wochenschrift, 1931.
37. "Contributo Allo Studio del Lobo Accessorio Della Vena Azigos," by Rene Jeanneret, M.D., Revista di Pathologia e Clinica della Tubercolosi, Anno V.
38. "Note Sur L'Operabilite des Adherences Apico-Mediastinales et Sur Leur Role dans L'Inefficacite du Pneumothorax," by Rene Jeanneret, M.D., and Victor Keser, M.D., Journal Medical de Leysin, July-August, 1939.
39. "Les Livres et la Lecture dans les Etablissements de Cure Pour Tuberculeux," by Rene Jeanneret, M.D., Revue Medicale de la Suisse Romande, September 1938.
40. "A Propos du Pronostic des Pleuresies Exsudatives," by Rene Jeanneret, M.D., and Andre Vaquette, Revue Medicale de la Suisse Romande, June 25, 1938.
41. "Pneumothorax Therapeutique et Epilepsie," by Rene Jeanneret, M.D., and A. Vaquette, M.D., Journal Medical de Leysin, September-October 1937.
42. "Faits Cliniques et Anatomiques," by Rene Jeanneret, M.D., and Willy Froehlich, M.D., Journal Medical de Leysin, December 1932.
43. "A Propos de la Phrenicectomie dans le Traitement de la Tuberculose Pulmonaire," by Pigue, Jeanneret, Froehlich, Journal Medical de Leysin, February 1931.
44. "A Propos des Corps Libres Intra-Pleuraux," by Piguet, and Jeanneret, Bulletin Medical de Leysin, February 1930.
45. "Granulation Tuberculeuse," presentation d'un document radiologique by Rene Jeanneret, M.D.
46. "Incidencia da Infeccao Tuberculosa Nos Universitarios Do Rio de Janeiro," by Drs. A. MacDowell, Reginaldo Fernandes, L. Arantes de Almeida, J. M. Castello Branco, Roberto Pereira, Olympio Gomes, Cesar de Almeida, Arnaldo de Barros, A. MacDowell Filho, Carlos Saboia, Affonso Bernardinelli Tarantino e Epilogo de Campos.
47. "Pneumotorax espontaneo com enfisema do mediastino," by Affonso MacDowell Filho.
48. "Pneumotorax hipertensivo na sinfise frouxa post-reexpansao aguda," by Affonso MacDowell Filho.
49. "Pneumotorax de reinstalacao," by Dr. Affonso MacDowell Filho.
50. "Contribuicao ao estudo da patogenia de B.C.G. vaselinado no coelho," by Arlindo de Assis e Amadeu Fialho.
51. "Tolerancia do organismo adulto-alergico ao B.C.G.," by Arlindo de Assis.
52. "Ensinamentos de dezessete anos (1927-44) de vacinacao B.C.G. no Brasil," by Arlindo de Assis.
53. "Sobre a estabilidade alergizante da orovacinacao B.C.G. no homem," by Arlindo de Assis de Carvalho.
54. "A proposito da transformacao dos tipos do bacilo de Koch (estudo da amostra humana 'Ratti,' do Instituto Pasteur de Paris)," by Arlindo de Assis.
55. "Estudos sobre alergia infratuberculinica III. Investigacoes em criancas imunicadas com B.C.G. por via oral," by Arlindo de Assis.
56. "Estudos sobre alergia infratuberculinica IV. Sua estimulacao especifica por via digestiva," by Arlindo de Assis e Wiberto Guedes-Pereira.
57. "Alguns aspectos novos da alergia tuberculinica nos analergicos vacinados com B.C.G. por via bucal," by Arlindo de Assis.
58. "Vacinacao B.C.G. por via bucal e alergia tuberculinica," by Arlindo de Assis e Alvimar de Carvalho.
59. "Sobre as imagens anormais do mediastino superior em criancas vacinadas com B.C.G. e vivendo em meio bacilifero," by Alvimar de Carvalho.
60. "Classificacao das Formas Clinicas da Tuberculose Pulmonar," by Alberto Renzo, Aloisyo de Paula.
61. "A Luta Contra a Tuberculose no Rio de Janeiro (O Departamento: Sua Organizacao)," Alberto Renzo e Jorge Marcellino Pinto Filho.
62. "Saude e Assistencia, Servico de Informacao Sanitaria," from Prof. Samuel Libanio.
63. "Inicio e Evolucao da Tuberculose Pulmonar do Adulto," by A. Ibiapina.
64. "Anais da 1a. Semana Brasileira Antituberculosa e 3a. Conferencia Regional de Tuberculose, Promovidas pela Sociedade Brasileira de Tuberculose," from Professor Antonio Ibiapina.
65. "Estudos Anatomo-Clinicos do Processo Tuberculoso," by Flavio Poppe de Figueiredo.

DISEASES OF THE CHEST

AUTHOR INDEX

Volume XIII, January - December, 1947

Adams, Ralph and Frank N. Allan. Thymectomy in the Treatment of Myasthenia Gravis. Discussion by F. P. O'Hara 436

Alarcon, Donato G. Penicillin in Acute Suppurations of the Lung 211

Andosca, John B. and John A. Foley. Intrapleural Pneumonolysis 248

Banyai, Andrew L. Fifteen Years' Experience with Carbon Dioxide in the Management of Cough 1

Banyai, A. L. Pneumoperitoneum Treatment of Pulmonary Tuberculosis. Editorial 387

Banyai, A. L. The Thirteenth Annual Meeting. Editorial 162

Barach, Alvan L. and Bettina Garthwaite. With the technical assistance of Eva Levenson and Dora Rader. Physiologic and Antibiotic (Penicillin) Therapy in Chronic Hypertrophic Pulmonary Emphysema 91

Bayliss, C. G. Closed Intrapleural Pneumonolysis 479

Bobrowitz, I. D. Treatment of Tension Cavities with Pneumothorax. Discussion by Benjamin L. Brock 133

Breckler, I. Alfred and Edwin R. Levine. A Technic for the Automatic Maintenance of Constant, Controlled Pneumothorax (A preliminary report) 698

Brock, Benjamin L. and Bernard D. Daitz. Work Capacity: Its Role in the Treatment of Tuberculosis 602

Burns, H. A. The Significance of the Time Element in Tuberculous Infiltrations. Discussions by H. I. Spector and J. A. Myers 456

Carr, Duane, W. E. Denman and E. F. Skinner. Noxious Gases and Bronchiectasis 596

Coulter, W. W. Jr. and Sam E. Thompson. A Plea for Increased Caution in the Use of Surgical Collapse Therapy for Pulmonary Tuberculosis 237

Duncan, Robert D. and Wayne W. Carpenter. Unilobar Tuberculosis Treated by Lobectomy 636

Freedlander, B. L. and F. A. French. Dihydrostreptomycin in Experimental Tuberculosis 708

Gibbons, Morton R. Jr. Treatment of Complications Arising in the Course of Pneumothorax Therapy of Pulmonary Tuberculosis. Discussions by John Roberts Phillips and Joseph Rosenblatt 673

Gilmore, David J. Pneumoperitoneum with Diaphragmatic Paralysis in the Treatment of Pulmonary Tuberculosis 153

Gines, Angel R. Surveys, BCG, Social Insurance and Health Card as Part of the Tuberculosis Control in Asuncion, Paraguay 20

Goldman, Alfred. The Surgical Treatment of Bronchial Adenoma. Discussion by J. J. Singer 321

Hartnett, W. G. and C. S. Beatty. An Unusual Aortic Aneurism (Followed for ten years) 627

Hendricks, Charles M. Medical Research. Editorial 273

Hendricks, Charles M. President's Address 391

Hetherington, L. H. and G. E. Spencer. Treatment of Recurrent Spontaneous Pneumothorax with Gomenol 652

Hochberg, Lew A., Israel G. Epstein and Morris Pernikoff. Endothelioma (Mesothelioma) of the Pleura (Presentation of a case treated surgically) 621

Hurst, Allan, H. M. Maier and Ralph Dwork. A Critical Study of Pneumoperitoneum and Phrenic Nerve Crush in Pulmonary Tuberculosis. Discussions by Harold W. Kohl, O. C. Schlack and Harold Guyon Trimble 345

Kettelkamp, George D. and Bernard Friedman. Diasone Therapy in Pulmonary Tuberculosis 23

Kinney, William M. Bronchiectasis: A Neglected Disease 33

Korol, Ephraim. Observations on Cystic and Bullous Emphysema of the Lungs (A study of 100 cases) 669

Kuzma, J. F. Histoplasmosis — The Pathologic and Clinical Findings 338

Levine, Edwin R. Inhalation Therapy in Chronic Bronchial Infections. Discussions by James S. Edlin and Arthur M. Olsen 295

Loeches, Hernandez. Treatment of Bronchiectasis and of Bronchial Suppurations by Intratracheal Injections of Penicillin. (Letter from Republic of Panama) 533

MacMillan, Angus E. Heparin in Empyema (Report of a case) 686

Marietta, Major General Shelley U. Installed as College President 397

Mark, Hilbert. Proposed State Tuberculosis Control Program 48

Mendenhall, Elliott. The Medical Management of the Bronchiectatic Patient 59

Meyer, W. L. Oleotherox 467

Movitt, E. R., J. V. Smith and Leo Eloesser. Treatment of Spontaneous Pneumothorax 221

Murphy, J. D. Thoracoscopy and Pneumonolysis 631

Myers, J. A. The College Research Program. Editorial 69

Myers, J. A. Community Tuberculosis Control. Editorial 261

Myers, J. A. Kendall Emerson Retires. Editorial 711

Myers, J. A. James E. Perkins Accepts. Editorial 713

Myers, J. A. The Pan-American Congress on Tuberculosis. Editorial 276

Myers, J. A. Tuberculosis in Hospital Patients. Editorial 385

Nixon, James W. Patent Ductus Arteriosus; Present Surgical Status. Discussion by O. C. Brantigan 360

Nixon, James W. and Joseph T. Goetz. The Second Stage Thoracoplasty 663

Nora, Ernest D. and Richard M. Davison. Pleurisy with Effusion Associated with Pseudomucinous Cystadenoma (Meig's Syndrome) 423

O'Rear, H. M. Hazards of Bovine Tuberculosis as a Matter of Public Health Significance and Potential Human Lung Infections with Bovine Tubercle Bacilli. Discussions by K. H. Pfuetze and J. A. Myers 368

Overholt, Richard H., Reeve H. Betts and Francis M. Woods. Multiple Segmental Resection in the Treatment of Bronchiectasis. Discussion by Frank P. Coleman 583

Poppe, J. K. The Diagnostic Significance of Clubbed Fingers 658

Quisumbing, Manuel, Sr. The Eradication of Tuberculosis; The Greatest Health Problem in the Philippines 516

Randolph, Howell and H. L. McMartin. Coccidioidomycosis in Phoenix, Arizona 471

Sauer, E. P. Conservative Management of Hemothorax with Presentation of an Illustrative Case 253

Scatchard, G. Newton and Diana Olga Duszynski. Routine Chest Roentgenograms of Hospital Admissions. Discussion by Donald McKay 312

Sherwood, J. Vincent. Tuberculosis Program in Grand Rapids, Mich. ... 202

Smith, Howard E. The Need for Tuberculosis Committees in County Medical Societies ... 528

Squier, Theodore L. Transitory Pulmonary Infiltration (Loeffler's Syndrome) ... 609

Strong, G. F. Cor Pulmonale ... 689

Titche, Leon L. Anesthesia in Tracheo-Bronchial Procedures ... 452

Tonolla, E. H. and O. C. Brantigan. Treatment of Thoracic Empyema with Sodium Tetradecyl Sulfate 1:500 in Azochloramid 1:3300 ... 123

Valle, Anibal Roberto and M. Lawrence White Jr. Subdiaphragmatic Aberrant Pulmonary Tissue (Case report) ... 63

Wolford, Roy A. The Tuberculous Veteran, Plans for His Future Care. Discussions by Arnold Shamaskin and Forrest G. Bell ... 189

DISEASES OF THE CHEST

SUBJECT INDEX

Volume XIII. January - December. 1947

— A —

Anesthesia in Tracheo-Bronchial Procedures. *Leon L. Titche* 452

Aneurism, Aortic. *W. G. Hartnett and C. S. Beatty* 627

Annual Meeting, American College of Chest Physicians 534
Executive Council 534
Board of Regents 534
Reports of Councils and Committees 534
Reports of Councils and Committees 715
Board of Governors 562
Council and Committee Appointments 563

— B —

Book Reviews:
Diseases of the Chest: With Emphasis on X-Ray Diagnosis. *By Eli H. Rubin* 734
Clinical Electrocardiography. *By David Scherf and Linn J. Boyd* 292
Principles in Roentgen Study of the Chest. *By William Snow* 293
Pulmonary Tuberculosis. *By Francis Kovats* 736
Pulmonary Tuberculosis in the Adult; Its Fundamental Aspects. *By Max Pinner* 292
The Lung. Second Edition. *By William Snow Miller* 293

Bovine Tuberculosis, Hazards of—. *H. M. O'Rear*. Discussions by Karl H. Pfuetze and J. A. Myers 368

Bronchial Adenoma, Surgical Treatment. *Alfred Goldman*. Discussion by J. J. Singer 321

Bronchial Infections, Chronic. Inhalation Therapy. *Edwin R. Levine*. Discussions by James S. Edlin and Arthur M. Olsen 295

Bronchiectasis: A Neglected Disease. *William M. Kinney* 33

Bronchiectasis, Medical Management. *Elliott Mendenhall* 59

Bronchiectasis and Noxious Gases. *Duane Carr, W. E. Denman and E. F. Skinner* 596

Bronchiectasis, Treatment of—. *Richard H. Overholt, Reeve H. Betts and F. M. Woods*. Discussion by Frank P. Coleman 583

— C —

Closed Intrapleural Pneumonolysis. *C. G. Bayliss* 479

Clubbed Fingers, Significance of—. *J. K. Poppe* 658

Coccidioidomycosis in Phoenix, Arizona. *Howell Randolph and H. L. McMartin* 471

College Research Program. Edit. 69

Community Tuberculosis Control. Edit. 261

Conservative Management of Hemothorax with Presentation of an Illustrative Case. *E. P. Sauer* 253

Cor Pulmonale. *G. F. Strong* 689

Cough, Management of—. *Andrew L. Banyai* 1

Critical Study of Pneumoperitoneum and Phrenic Nerve Crush in Pulmonary Tuberculosis. *Allan Hurst, H. M. Maier and Ralph Dwork*. Discussions by Harold W. Kohl, O. C. Schlack and Harold G. Trimble 345

— D —

Diagnostic Significance of Clubbed Fingers. *J. K. Poppe* 658

Diasone Therapy in Pulmonary Tuberculosis. *George D. Kettelkamp and Bernard Friedman* 23

Dihydrostreptomycin in Experimental Tuberculosis. *B. L. Freedlander and F. A. French* 708

—E—

Emphysema, Cystic and Bullous—. *Ephraim Korol* 669

Emphysema, Physiologic and Antibiotic (Penicillin) Therapy—. *A. L. Barach, et al* 91

Empyema, Heparin in—. *Angus E. MacMillan* 686

Empyema, Treatment of—. *E. H. Tonolla and O. C. Brantigan* 123

Endothelioma (Mesothelioma) of the Pleura (Presentation of a case treated surgically). *Lew A. Hochberg, Israel G. Epstein and Morris Pernikoff* 621

Eradication of Tuberculosis; The Greatest Health Problem in the Philippines. *Manuel Quisumbing Sr.* 516

—F—

Fifteen Years' Experience with Carbon Dioxide in the Management of Cough. *Andrew L. Banyai* 1

—H—

Hazards of Bovine Tuberculosis as a Matter of Public Health Significance and Potential Human Lung Infections with Bovine Tubercle Bacilli. *H. M. O'Rear.* Discussions by K. H. Pfuetze and J. A. Myers 368

Hemothorax. *E. P. Sauer* 253

Heparin in Empyema (Report of a Case). *Angus E. MacMillan* 686

Histoplasmosis—The Pathologic and Clinical Findings. *J. F. Kuzma* 338

—I—

"Information Please in Medicine" (As Presented at the Twelfth Annual Meeting of the American College of Chest Physicians, San Francisco, June 29, 1947) 263

Inhalation Therapy in Chronic Bronchial Infections. *Edwin R. Levine.* Discussions by James S. Edlin and Arthur M. Olsen 295

Intrapleural Pneumonolysis. *John B. Andosca and John A. Foley* 248

—J—

James E. Perkins Accepts. Edit. 713

—K—

Kendall Emerson Retires. Edit. 711

—L—

Lobectomy in Unilobar Tuberculosis. *Robert D. Duncan and Wayne W. Carpenter* 636

—M—

Medical Management of the Bronchiectatic Patient. *Elliott Mendenhall* 59

Medical Research. Edit. 273

Multiple Segmental Resection in the Treatment of Bronchiectasis. *Richard H. Overholt, Reeve H. Betts and F. M. Woods.* Discussion by Frank P. Coleman 583

Myasthenia Gravis, Treatment of—. *Ralph Adams and Frank N. Allan* 436

—N—

Need for Tuberculosis Committees in County Medical Societies. *Howard E. Smith* 528

Noxious Gases and Bronchiectasis. *Duane Carr, W. E. Denman and E. F. Skinner* 596

—O—

Obituaries:

Agar, John S., Jr. 87

Clark, William S. III 185

Donnelly, John 738

Ferrell, Lee T. 737

Fish, Clyde M. 87

Ghosh, Jaharlal 739

King, Drue 737

Nelson, Ira D. 739

Watterson, Walter H. 738

Yellin, Daniel 294

Observations on Cystic and Bullous Emphysema of the Lungs. (A Study of 100 Cases). *Ephraim Korol* 669

Oleothorax. *W. L. Meyer* 467

—P—

Pan-American Congress on Tuberculosis 277

Pan-American Congress on Tuberculosis. Edit. 276

Paraguay, Tuberculosis Control—. *Angel R. Gines* 20

Patent Ductus Arteriosus; Present Surgical Status. *James W. Nixon.* Discussion by O. C. Brantigan 360

Penicillin in Acute Suppurations of the Lung. *Donato G. Alarcon* 211

Philippines, Tuberculosis Problem—. *Manuel Quisumbing, Sr.* 516

Phrenic Nerve Crush in Pulmonary Tuberculosis—. *Allan Hurst, H. M. Maier and Ralph Dwork* 345

Physiologic and Antibiotic (Penicillin) Therapy in Chronic Hypertrophic Pulmonary Emphysema. *Alvan L. Barach and Bettina Garthwaite* 91

Plea for Increased Caution in the Use of Surgical Collapse Therapy for Pulmonary Tuberculosis. *W. W. Coulter, Jr. and Sam E. Thompson* 237

Pleurisy with Effusion Associated with Pseudomucinous Cystadenoma (Meig's Syndrome). *Ernest D. Nora and Richard M. Davison* 423

Pneumonolysis and Thoracoscopy. *J. D. Murphy* 631

Pneumonolysis, Closed Intrapleural. *C. G. Bayliss* 479

Pneumonolysis, Intrapleural. *J. B. Andosca and J. A. Foley* 248

Pneumoperitoneum with Diaphragmatic Paralysis in the Treatment of Pulmonary Tuberculosis. *David J. Gilmore* 153

Pneumoperitoneum and Phrenic Nerve Crush in Pulmonary Tuberculosis. *Allan Hurst, H. M. Maier and Ralph Dwork* 345

Pneumoperitoneum Treatment of Pulmonary Tuberculosis. Edit. 387

Pneumothorax. *I. D. Bobrowitz* 133

Pneumothorax. *I. Alfred Breckler and Edwin R. Levine* 698

Pneumothorax. *M. R. Gibbons, Jr.* 673

Pneumothorax, Spontaneous—. *E. R. Movitt, J. V. Smith and Leo Eloesser* 221

Pneumothorax, Spontaneous. Treatment with Gomenol. *L. H. Hetherington and G. E. Spencer* 652

Proposed State Tuberculosis Control Program. *Hilbert Mark* 48

Retiring President's Address. *Charles M. Hendricks* 391

—R—

Routine Chest Roentgenograms of Hospital Admissions. *G. Newton Scatchard and Diana Olga Duszynski.* Discussion by Donald McKay 312

—S—

Second Stage Thoracoplasty. *James W. Nixon and Joseph T. Goetz* 663

Significance of the Time Element in Tuberculous Infiltrations. *H. A. Burns.* Discussions by H. I. Spector and J. A. Myers 456

Streptomycin in Tuberculosis. Report of Committee on Chemotherapy and Allied Measures 169

Subdiaphragmatic Aberrant Pulmonary Tissue (Case Report). *Anibal Roberto Valle and M. Lawrence White, Jr.* 63

Surgical Treatment of Bronchial Adenoma. *Alfred Goldman.* Discussion by J. J. Singer 321

Surveys, BCG, Social Insurance and Health Card as Part of the Tuberculosis Control in Asuncion, Paraguay. *Angel R. Gines* 20

— T —

Technic for the Automatic Maintenance of Constant, Controlled Pneumothorax. (A Preliminary Report). *I. Alfred Breckler and Edwin R. Levine* 698

Thirteenth Annual Meeting. Edit. 162

Thoracoplasty, Second Stage. *J. W. Nixon and J. T. Goetz* 663

Thoracoplasty and Pneumonolysis. *J. D. Murphy* 631

Thymectomy in the Treatment of Myasthenia Gravis. *Ralph Adams and Frank N. Allan*. Discussion by F. P. O'Hara 436

Transitory Pulmonary Infiltration (Loeffler's Syndrome). *Theodore L. Squier* 609

Treatment of Bronchiectasis and of Bronchial Suppurations by Intratracheal Injections of Penicillin. *Hernandez Loeches* 533

Treatment of Complications Arising in the Course of Pneumothorax Therapy of Pulmonary Tuberculosis. *Morton R. Gibbons, Jr.* Discussions by John Roberts Phillips and Joseph Rosenblatt 673

Treatment of Recurrent Spontaneous Pneumothorax with Gomenol. *L. H. Hetherington and G. E. Spencer* 652

Treatment of Spontaneous Pneumothorax. *E. R. Movitt, J. V. Smith and Leo Eloesser* 221

Treatment of Tension Cavities with Pneumothorax. *I. D. Bobrowitz*. Discussion by Benjamin L. Brock 133

Treatment of Thoracic Empyema with Sodium Tetradecyl Sulfate 1:500 in Azochloramid 1:3300. *E. H. Tonolla and O. C. Brantigan* 123

Tuberculosis, Diasone Therapy. *G. D. Kettelkamp and Bernard Friedman* 23

Tuberculosis in Hospital Patients. Edit. 385

Tuberculous Infiltrations, Time Element. *H. A. Burns* 456

Tuberculosis, Pneumoperitoneum Treatment—. *D. J. Gilmore* 153

Tuberculosis Program in Grand Rapids, Michigan. *J. Vincent Sherwood* 202

Tuberculosis, Study of Pneumoperitoneum and Phrenic Nerve Crush. *Allan Hurst, H. M. Maier and Ralph Dwork* 345

Tuberculosis, Surgical Collapse Therapy—. *W. W. Coulter, Jr. and S. E. Thompson* 237

Tuberculosis, Unilobar, Treated by Lobectomy. *R. D. Duncan and W. W. Carpenter* 636

Tuberculosis, Work Capacity—. *Benjamin L. Brock and Bernard D. Daitz* 602

Tuberculous Veteran, Plans for His Future Care. *Roy A. Wolford*. Discussion by Arnold Shamaskin and Forrest G. Bell 189

— U —

Unilobar Tuberculosis Treated by Lobectomy. *Robert D. Duncan and Wayne W. Carpenter* 636

Unusual Aortic Aneurism (Followed for Ten Years). *W. G. Hartnett and C. S. Beatty* 627

— W —

Work Capacity: Its Role in the Treatment of Tuberculosis. *Benjamin L. Brock and Bernard D. Daitz* 602

DISEASES *of the* CHEST

OFFICIAL JOURNAL OF THE

AMERICAN COLLEGE OF CHEST PHYSICIANS

EDITORIAL OFFICE
111 Millard Hall
University of Minnesota
Minneapolis 14, Minnesota

EXECUTIVE OFFICE
500 North Dearborn Street
Chicago 10, Illinois

DISEASES OF THE CHEST is published bi-monthly by the American College of Chest Physicians. The contents of the journal consist of contributions in the field of diseases of the chest, book reviews, editorials, case reports, news items and matters concerning the affairs of the American College of Chest Physicians and its affiliated societies.

GENERAL INFORMATION

ORIGINAL ARTICLES only are published with the understanding that they are contributed exclusively to this Journal. Manuscripts offered for publication, correspondence relating to the editorial management and books for review should be sent to The Editor, *Diseases of the Chest*, 111 Millard Hall, University of Minnesota, Minneapolis 14, Minn. The publishers are not responsible for statements made or opinions expressed by contributors in articles published in its columns. Manuscripts are subject to editorial modification.

CUTS FOR ILLUSTRATIONS, DRAWINGS and CHARTS will be supplied without charge in moderate number, but special arrangements must be made with the editor and publishers for excess illustrations and elaborate tables.

REPRINTS are furnished on order only and must be requested of the publishers when galley proofs are submitted. Prices will be quoted at that time.

COPYRIGHTS cover publication in *Diseases of the Chest* and articles may not be reproduced without permission of the publishers.

BUSINESS CORRESPONDENCE regarding subscriptions, advertisements, and other business of *Diseases of the Chest* should be addressed to the American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

CHANGES OF ADDRESS NOTICES should include the old as well as the new address.

PREPARATION OF MANUSCRIPTS

MANUSCRIPTS must be typewritten, double spaced, with good margins, on one side of the paper only. Send original copy to the editor, retaining a carbon.

DRAWINGS AND CHARTS must be made with BLACK INK to permit of best reproduction. Photographic prints of plates or slides on glossy paper produce the best halftones. Write the number of each illustration, drawing or chart on the back thereof together with the author's name and abbreviated title of the article.

LEGENDS FOR ILLUSTRATIONS, etc.: Typewrite list of same at end of manuscript with reference to number of illustration, drawing or chart.

BIBLIOGRAPHIES: Prepare carefully and fully to avoid confusion. Include in each reference (1) Number, (2) Author's last name followed by initials, (3) Title of article, (4) Name of periodical or book, (5) Volume, page and year if a periodical OR publisher if a book, viz.: 1 Clerf, L. H.: "Tuberculous Tracheobronchitis," *Dis. of Chest*, VIII: 11, 1942.

Place list of references at end of article, not in footnotes. Arrange in numerical order.

A brief summary should be included for translation into Spanish.

FULL ADDRESS of author should appear somewhere on manuscript, preferably at the end.

ANNUAL SUBSCRIPTION

United States of America $5.00 **Foreign Countries $6.00**

Mead's Standardized Cod Liver Oil

•

- High potency
- Biologically assayed
- Fine flavor
- Uniform vitamin content
- Low in fatty acids
- Council accepted
- Not advertised to the laity

•

Samples and literature sent on request to physicians. Please enclose professional card to cooperate in preventing samples reaching unauthorized persons.

•

MEAD JOHNSON & COMPANY

Evansville, Indiana, U.S.A.